ARCHIVES OF

NEUROLOGY and PSYCHIATRY

CONTENTS OF PREVIOUS NUMBERS

JULY, 1924. NUMBER 1

Myelinogeny as Applied to the Study of Behavior. Frederick Tilney, M.D., and Louis Casamajor, M.D., New York.

Trypanamide Penetration into the Central Nervous Tissue With and Without Spinal Irritation; Comparison with Arsphenamin. H. G. Mehrtens, M.D., Frank Koloc and Helen Marshall, M.A., San Francisco.

Nonspecific Stimulation Therapy in Tabes Dorsalis. Edward H. Ahlswede, M.D., Buffalo.

Herpes Zoster; Central Nervous System Lesions Similar to Those of Epidemic (Lethargic) Encephalitis: Report of a Case. William Thalhimer, M.D., Milwaukee.

Abstracts from Current Literature.

Society Transactions:
Philadelphia Neurological Society.
New York Neurological Society.
Boston Society of Psychiatry and Neurology.

Book Reviews.

AUGUST, 1924. NUMBER 2

The So-Called Unit Characters in Relation to Hereditary Disturbances of the Nervous System. Walter Timme, M.D., New York.

End Results in Sixty-Two Cases of Spina Bifida and Cephalocele. George David Cutler, M.D., Boston.

A Developmental Anomaly of the Cerebral Cortex. Eric A. Linell, M.D., and Basil McB. Koster, M.D., Toronto, Canada.

Localized Meningitis with the Syndrome of Froin in the Spinal Fluid. Norman L. Clarke, M.D., Ann Arbor, Mich.

A Comparative Study of Syphilis in Colored and in White Women with Mental Disorder. L. D. Hubbard, M.D., Washington, D. C.

Hereditary Factors Responsible for Development of Optic Atrophy and Retinitis Pigmentosa. W. H. Wilmer, M.D., Washington, D. C.

Pathology and Pathogenesis of Unilateral Internal Hydrocephalus: Report of a Case. F. W. Winkelman, M.D., Philadelphia, and John L. Eckel, M.D., Buffalo.

News and Comment: The American Psychiatric Association.

Abstracts from Current Literature.

Society Transactions:
Philadelphia Neurological Society.
Chicago Neurological Society.

Book Review.

SEPTEMBER, 1924. NUMBER 3

Studies of Cerebral Function in Learning. V. The Retention of Motor Habits After Destruction of the So-Called Motor Areas in Primates. K. S. Lashley, Ph.D., Minneapolis.

Tumor of the Hypophysis: Report of a Case. James Hendrie Lloyd, M.D., and Francis C. Grant, M.D., Philadelphia.

The Significance of Certain Factors for the Development of the Heredofamilial Diseases of the Nervous System (Consanguinity, Race, Endocrinopathies, Toxins, Infection, Alcoholism, Roentgen Rays). Lewellys F. Barker, M.D., Baltimore.

Studies in Decerebration. II. An Acute Decerebrate Preparation. Lewis J. Pollock, M.D., and Loyal E. Davis, M.D., Chicago.

Clinical and Occasional Notes:
Apparatus for Simultaneous Displacement. Frank Liberson, M.D., New York.
Anterognosia. Foster Kennedy, M.D., New York.

Abstracts from Current Literature.

Society Transactions:
Boston Society of Psychiatry and Neurology.
Philadelphia Neurological Society.

Book Reviews.

COPYRIGHT, 1924, BY THE AMERICAN MEDICAL ASSOCIATION

TREATMENT OF GENERAL PARALYSIS

RESULTS OBTAINED IN A SERIES OF FIVE HUNDRED CASES [*]

I. J. FURMAN, M.D.

Senior Assistant Physician, Manhattan State Hospital

WARD'S ISLAND, N. Y.

In October, 1919, a definite plan for the treatment of patients with neurosyphilis was adopted at the Manhattan State Hospital, with the intention of treating a large number for a sufficient length of time to obtain data on which to base reliable conclusions concerning the results of treatment.

Because of the irregularity in the progress of the parenchymatous form of neurosyphilis in different persons, it was fully appreciated at the outset that a large number of patients must be treated for a time longer than the ordinary period of remissions in untreated cases, in order to be certain that the periods of improvement were due to treatment and not to the usual remissions. The patients accepted for treatment must not be selected but should be taken in the order of their admission, with the fewest possible exceptions, because a group of selected patients would not be representative of the general run of cases, either in or outside of the hospital. Also, it is admittedly unsafe to base an opinion on the results obtained in a few cases, even though they are taken in the order of admission, for, in a small number, incidental factors may influence the percentage ratio so easily that there is a great possibility of error. Further, if any special group were chosen, no comparable group of untreated cases would be available to contrast with it. However, if the patients selected for treatment are taken in the order of admission, it is possible to obtain a control by taking a sufficient number of syphilitic patients (also in the order of their admission, in this instance before October, 1919) before any treatment was started. This method of control has been used at this hospital,[1] and as these two classes, reported in this study, are identical in every respect except the treat-

---

[*] Read at the Inter-Hospital Conference, at Ward's Island, N. Y., June 6, 1923.

1. Raynor, M. W.: Remissions in General Paralysis: A Study of Consecutive Admissions of Men to the Manhattan State Hospital from July 1, 1911, to June 30, 1918, published in this issue of THE ARCHIVES, p. 419.

ment, it seems justifiable to ascribe any variation in the course, duration and outcome of the two groups to the only added factor, that is, treatment. More than 500 treated cases of paralysis have been used on which to formulate deductions, and these have all been in men.

In a previous report on this subject, in March, 1922, a detailed description of the order of selecting patients for treatment and the method of treatment, were given.[2] During the past fourteen months, there has been no change in the character of patients chosen for treatment, and there has also been little variation in the method of treatment employed, except that, before March, 1922, only twenty-five patients had been treated with neo-arsphenamin plus systematic spinal drainage, all the other patients receiving only the intravenous injections. Twenty-five patients not receiving spinal drainage, and used as controls, did not seem to benefit as much from treatment as did the twenty-five who received the combined treatment. The encouraging benefits of drainage in these cases seemed to indicate that all patients should be given this added procedure, and, accordingly, for the past fourteen months, drainage has been carried out systematically. Two other points in relation to treatment should be mentioned. The first concerns the dosage, and the second the length of a course of treatment. By careful preparation of the patient, and by observing certain precautions in the administration of the neo-arsphenamin, the untoward results of treatment have been reduced to a minimum, but still a few cases of dermatitis, anaphylaxis, stomatitis and especially the acute digestive disturbances, immediately following treatment, continued to appear.

In any disease, the form of treatment which produces the greatest good with the least amount of discomfort is the most desirable. This is especially true in mental cases, in which one disagreeable occurrence may destroy all future cooperation. After considerable observation, it was concluded that large initial doses of arsphenamin more often caused the immediate disagreeable untoward symptoms, such as anaphylaxis, nausea, vomiting, headache, chills, and other symptoms, than did the smaller doses, and that maximum doses given for any length of time often caused severe jaundice and the serious skin and mouth troubles which are invariably associated with marked secondary anemia and great destruction of the marrow of the long bones. For at least one year, the maximum dose of neo-arsphenamin has been 0.6 gm. until several treatments have been given, or until the exact tolerance of each patient has been determined, and there seems to be no safer way to discover this maximum tolerance point than to begin with a small dose and proceed cautiously toward the largest dose that can be absorbed

2. Treatment of Neurosyphilis at the Manhattan State Hospital, State Hosp. Quart **8**:84 (Nov.) 1922.

by the particular patient. Comparatively few patients are now regularly receiving more than 0.6 gm. While body weight is an aid in arriving at a suitable dose of arsphenamin, there are so many exceptions that it cannot be used as the sole criterion. It has been found that some large robust patients can take care of only small doses, while other small emaciated patients are able to receive maximum doses without unfavorable results; in other words, the individual idiosyncrasy is the most reliable key to the dosage. These, individually tested, medium-sized doses, are not only better tolerated at the time of administration, but they can be given over a longer period of time without serious cumulative results. Advantage has been taken of this fact by increasing the number of doses in a series from the ten weekly doses of neo-arsphenamin, followed by twelve of mercury, previously considered a series, to fifteen of arsphenamin, followed by fifteen of mercuric salicylate. A course so conducted consumes about eight months of continuous treatment. After a full course is completed, if there are no special indications requiring a continuation, a rest of three months is given before further treatment is resumed. By following this outline, a patient is under treatment about two thirds of the time and resting one third of the time. A few patients have been treated constantly for a whole year, because in previous series they did not seem to do as well when the treatment was stopped. From the results obtained in these and also from the results of longer courses, the impression is gradually gaining ground that uninterrupted treatment should perhaps be adopted as a routine method in all cases.

Our experience also indicates that spinal drainage is a valuable part of the treatment of cerebrospinal syphilis, and it is a question whether this should not be continued through the resting period in patients treated intermittently, for in a great majority of cases, the spinal fluid pressure is greatly reduced and remains so even at the end of a course. Therefore, if drainage was useful to reduce pressure during treatment, which seems to be true, then, it should be continued to control pressure between courses. The exact value of such continuous drainage cannot be stated at present, but in a certain number of cases this treatment will be given in the future and the results observed.

As far as can be ascertained, there seems to be no authentic reports showing the recovery of a patient with general paralysis, either spontaneously or as a result of treatment. This is not a sufficient reason, however, for abandoning our efforts, for the discovery of a cure in any disease has usually been the outcome of patient work and a hopeful attitude. Again, there are still many incurable diseases in which we are forced to use palliative measures, and even though this is understood, such relief would not be withheld. In neurosyphilis, it is often

impossible to differentiate clinically general paralysis and cerebral syph-
ilis, or general paralysis from a combination of the two, and often the
laboratory is unable to establish this difference by serologic examination.
This leaves only two other ways of solving this question: by necropsy
and by treatment. Obviously, the first is of no value to the patient;
therefore, the therapeutic method is the only remaining means of making
a more exact diagnosis. There is no apparent reason why such a method
of diagnosis should not be used in every suitable case of neurosyphilis,
and an intensive course of treatment given at once. The results of
such an initial course could then be used as an aid in deciding whether
we are dealing with a favorable form of cerebral syphilis or whether
we have an uncomplicated case of general paralysis, and should give
a less intensive, continuous course. The intensive treatment should be
continued with the hope of obtaining satisfactory improvement.

It might be argued that the therapeutic method is a poor one, which
no doubt is true, but as it is the only one available, it seems reasonable
to employ it until some better way is found, especially as it is, at
the same time, giving the patient the benefit of a recognized treatment
for syphilis; moreover, few of these patients have received any treat-
ment for years before coming to the hospital, and these have perhaps
had only a few doses of mercury or local applications to the chancre;
only a small percentage of the cases have ever been given arsenic in
any form, so why should it be condemned without a trial? The objec-
tion might also be raised that small doses of arsphenamin amount to
inadequate treatment, and so administered are liable to stimulate the
disease rather than to retard it. It is doubtful whether there have been
sufficient data accumulated to establish this opinion, and it can, no
doubt, be placed in the class with the objection to using arsphenamin in
the initial stage for fear that it will cause more cases of neurosyphilis.
The latter opinion seems to be largely refuted by the fact that so large a
percentage of syphilitic patients show spinal fluid changes during the
first few months of infection, and these early changes in the serology
seem to bear no close relationship to the form of treatment given;
further, if arsphenamin is a factor in causing syphilis to enter the central
nervous system, and small doses or inadequate treatment stimulates the
progress of cerebrospinal syphilis, it seems that it has now been in use
for a sufficient length of time to cause an increase in the number of
neurosyphilitic patients, and consequently an increase of general paraly-
sis, which is not true, if the admissions to this hospital can be used as
a criterion. The number of paretic patients admitted here has not
varied to any extent in several years.

Concerning the benefits derived from treatment, it is felt that a
more definite opinion can be expressed than at the time of the pre-

liminary report. Since then, a more extensive system of records has been kept, showing frequent observations of each patient, taking into account the physical, mental neurologic and serologic progress of each treated patient. These records show results which agree consistently with the results of treatment from the beginning of the study; about 60 per cent. show such marked physical improvement that they remain up and around, and require much less care, and many are quite useful. Table 5 shows the approximate proportion of idle and employed ambulatory patients remaining in the hospital, which indicates what might be called definite improvement in the hospital. This physical improvement continues to be demonstrated by the fewer patients with neurosyphilis, in bed. The number is only about 55 per cent. of the number before systematic treatment was started. About 50 per cent. of the treated patients show definite mental improvement, which is indicated by much less general confusion, fewer confused episodes, better general grasp and fewer frank convulsions. However, when convulsions do occur, they are more liable to be severe and prolonged and to terminate in death; frequently the first and only convulsion from which a patient suffers overtakes him suddenly when he is in good general health, and ends fatally within three or four days. The average time in bed for all treated patients dying in the hospital for the past two years, has been about fifteen days. There seems to be no doubt that rapid failure followed by sudden death is the rule in these treated patients, with relatively few dying from exhaustion after a long bedridden state. Neurologically, there have been no consistent changes noted which could be attributed to treatment. Serologically, there continues to be a marked reduction in the cytology in practically every case; usually the higher the cell count at the beginning of the treatment, the greater is the reduction. The globulin content varies in unison with the cells in most cases, but this is not always true, and it seems almost impossible to cause the globulin to disappear entirely. The Wassermann reaction on the blood is frequently reduced without a parallel reduction in the Wassermann reaction on the spinal fluid, and vice versa, but the blood is more often favorably affected than the spinal fluid. While the cytology and globulin have often improved without a corresponding improvement in the Wassermann test on the fluid or the blood, in no case has the Wassermann reaction on either the fluid or blood been reduced without a foregoing reduction in the other two phases. The globulin content and the cell count appear to indicate the more immediate well-being of the patient, for the records show, too frequently to be ignored, that a rapid rise in the globulin and cell count in the spinal fluid shows that the patient is approaching a critical period. These changes have been observed even at a time when the patient seemed to be

progressing very well, but, within a few days a severe seizure would develop, followed by a rapid decline and possibly death.

RESULTS OF TREATMENT FROM 1921 TO 1923

The figures in Table 1 are based on 503 treated cases of paralysis.

This table shows the proportion of treated patients compared with admissions, and also the number of treated and untreated patients as they have been admitted each year. It also shows the reasons why treatment was not given. The fifty patients treated before July, 1920, were not accepted in their order of admission and should not be considered, but have been included in order to complete the number of patients treated since 1920, which, taken up to May 1 of this year, amount to 503.

The number of treated patients during the past three years is shown to be quite consistently in proportion to the number of admissions,

TABLE 1.—*Reasons Not Treated*

Date	Total Admissions	Number Treated	Number Not Treated	Died too Soon	Too Weak	Discharged too Soon	Too Disturbed
1921*	...	50	0	0	0	0	0
1921	220	165	61	32	16	8	5
1922	230	162	62	36	13	5	8
1923†	182	126	56	34	12	6	4
Total	632	503	179	102	41	19	17

* Not taken in order of admission, and only mentioned to complete the whole number treated.
† Ten months, hospital year, ends June 30, 1923.

although 126 treated patients during the last ten months is rather low, and probably is due to a change in the character of cases admitted. Since January the proportion of stretcher cases admitted here has been much larger, and as a result there have been a smaller number of cases suitable for treatment.

Table 1 also shows that a large number of the untreated cases appear as such because death occurred shortly after admission. All of these patients died within one month after entering the hospital; many died within a few days; in others, there was some delay in arriving at a final diagnosis, so that treatment could not be started. A few of these patients have received two or three doses of neo-arsphenamin or mercury, but not enough treatment to exert any influence on the course of the disease, so they have not been counted.

The figures show nineteen patients discharged so soon that no treatment could be given; treatment would have been desirable for many of these, and no doubt would have benefited them.

In the three years, less than sixty patients have been eliminated indefinitely, because they have been constantly too weak or too disturbed

for treatment. This fact seems to show that by close observation of the patients' condition, few neurosyphilitic patients who remain in the hospital more than one month need to be excluded from treatment.

Table 2 shows the number of treated and untreated patients that have been paroled, and the duration of such parole.

Twenty-six patients were paroled in 1921, thirty-two in 1922 and thirty-four in the first ten months of 1923. At the present rate for this year, there will be forty patients paroled by the end of the fiscal year, or an increase of 50 per cent. over 1921, the first year that systematic treatment was carried out. It is felt that this gain indicates that the effects of treatment are gaining momentum. The figures show that more paroles were terminated by discharge at their expiration in 1921 than in either 1922 or 1923, but this is no doubt due to the fact that the great majority of paroles in 1921 were terminated at the end of six months, as that was the limit of most paroles at that time, while, during the last two years, most of these patients have gone out on a year's parole, and

TABLE 2.—*Number of Patients Paroled and Duration of Paroles*

Date of Admission	Paroled	Duration of Paroles					Discharged at End of Parole	Returned	Remained on Parole	Untreated Patients Paroled
		1 Mo.	1-3 Mo.	3-6 Mo.	6-12 Mo.	Total				
1921..........	26	2	3	4	23	32	25	7	9	3
					6-18 mo.					
1922..........	32	7	1	3	10	21	6	15	19	4
1923 (10 mo.)	34	0	4	1	24	29	19	10	19	3
	92	9	8	8	57	82	50	32	47	10

in many cases the time has been extended to eighteen months or even two years.

Nineteen patients discharged at the expiration of their parole during the first ten months of this year indicate that the discharges this year will about equal those of 1921. This means, however, that the collective number of days of parole will be much greater for 1923 than 1921, by reason of the greater length of individual paroles. Although there were more discharged in 1921, there were also fewer remaining on parole than in either year since. While the longer paroles present more opportunity for patients to be returned and not discharged, it also has a tendency to increase the number remaining on parole at the end of the year.

The change in the parole period has caused this table to show confusing results, but has had no influence on the actual number paroled, which is showing a gradual increase.

Of the twenty-six patients paroled in 1921, three received no treatment; one was returned during the first month; one was returned in the third month, and the third was discharged at the expiration of a six months' parole in only fair condition. He died at home two months later.

Of the thirty-two patients going on parole in 1922, there were four untreated. Three of these were returned during the third month and one in the fifth—all in poor condition. The thirty-four paroled for the ten months of this year include three untreated patients, and all have remained out until the present time, but none of them has been out over five months, and all are only slightly improved. It is realized that these untreated patients for some reason were unfavorable for treatment, but that they did improve sufficiently to be paroled; however, the fact is evident that the great majority of paroles of neurosyphilitic patients come from those treated and that these are also the ones remaining out and finally discharged.

TABLE 3.—*Living Treated Patients*

Before	Number Treated	Still Alive	Still Alive in Hospital	Still Alive Out of Hospital
1920	50	11	9	2
1921	165	41	36	5
1922	162	62	45	17
1923 (10 months)	126	84	65	19
	503	198	155	43

This table indicates the proportion of patients living after one, two and three years of treatment; also the proportion of those living inside and outside of the hospital. Of a total of 198 living patients, there are forty-three living outside the institution, a fairly large proportion, and

TABLE 4.—*Condition of Paroled Patients*

	Paroled	Treated	Remaining on Parole	Living	True Remissions	Paroled Improved	Paroled Unimproved	Untreated
1921	26	23	0	5	4	8	11	3
1922	32	27	4	17	6	12	9	5
1923 (10 months)	34	29	15	19	9	14	6	5
	92	79	19	41	19	34	26	13

more than in previous years. This seems to show that in a general way there has been some added factor which was not present before and which is causing sufficient improvement to merit attention; we feel justified in attributing this improvement to treatment given both while in the hospital and while on parole, and, in some cases, after discharge. This extramural advice and treatment are considered an essential part of the regimen, and it is gratifying to see how willingly practically all patients return for treatment.

Table 4 shows especially the number of true remissions occurring since 1920 in patients that have been paroled.

It might be well to mention the conditions required for a true remission: First, the patient must be in good general physical health, be

free from psychotic symptoms, and possess insight; the neurologic symptoms, such as changes in the deep reflexes and pupils, are so constantly present and seem to bear so little relationship to the physical and mental health that a disappearance or even improvement of these symptoms has not been required. Second, the patient must be able to go outside and engage in some profitable employment away from home, and show no disorder of conduct or behavior. In these improved patients, the cell count has always been reduced, the globulin changed for the better, and the Wassermann reactions on both blood and fluid have shown variable results, some showing a decrease and some remaining stationary.

Accepting the foregoing criteria for remissions, there were four such cases in 1921, six in 1922 and nine so far this year; there are also two of the first fifty patients treated still living and in good remission, being not only self-supporting, but also helping to care for their families. These two patients have either come back to the hospital for treatment, or have been given arsphenamin in a city clinic in regular courses. Of the four considered in a true remission of the 1921 group, one died after being out of the hospital nineteen months; his death was sudden, following a series of convulsions, lasting four days; he was working as a salesman, and was taken ill while employed. The other three patients are still employed. The six in the 1922 group are still living; five are working away from home, but the sixth has recently had to give up work and remain at home because of ataxia; he has resumed treatment, which he had neglected for several months following his discharge. Of the nine patients enjoying remissions in 1923, one has been returned to the hospital because of a period of excitement following employment as a salesman for seven months. The other eight are fulfilling the requirements of a true remission. A few of the patients paroled as improved, also shown in Table 4, have continued to improve at home, but none of them has appeared to conform to a case of true remission for any length of time, and therefore they are not considered as having true remissions. They are able to work at home, however, and show no conduct disorder, but they do not possess the ability to take up work outside and meet the competition that such employment demands. Of the thirteen untreated patients on parole, none has shown anything like a true remission. These are only mentioned incidentally, for it is manifestly unfair to contrast this group with the others. They really represent some of the most unfavorable cases from the beginning. It seems that these remissions in treated patients have averaged longer than the ordinary remission without treatment, and the patients have returned more nearly to their normal condition.

Referring to Table 5, which shows the improvement in treated patients who have been continuously in the hospital, it may be said that by going over the records in all these cases, it was found that 60 per cent. of the patients were described as physically better and about 50 per cent. as mentally improved, so that we are becoming convinced that treated patients who never leave the hospital show both a physical and mental improvement. In Table 5 are given the actual figures showing such improvement during the last three years, and also the number of patients who have taken up useful occupations in the hospital. These have worked in the different departments of the institution, such as the mechanical, occupational, industrial, etc., and they have also done outside work around the grounds. A few have been counted as working in the ward, but they have been working in the clothing rooms and doing other work requiring thought and initiative, and have not been simply pushing a polisher for a short time each day, which is often called "ward work." An attempt has been made to estimate the duration of the life

TABLE 5.—*Treated Patients Showing Improvement Who Have Been in the Hospital Continuously*

Date	Treated	Physically Improved	Mentally Improved	Ambulatory	
				Employed	Idle
1921	165	90	82	46	53
1922	162	97	81	47	50
1923	126	75	63	35	40
	453	271	226	128	143

of treated patients, but this seems premature because the proportion still living is quite large and renders such a table practically without value at present.

CONCLUSIONS

From the work done at the Manhattan State Hospital, we feel that the most outstanding result of treatment of paralysis so far is the improvement in the general health of the patients. This is shown by the fact that fewer are confined in bed, that a greater number are paroled for a longer period and remain out of the hospital until the expiration of their parole, and that there are more true remissions.

We feel that every psychiatric hospital should adopt the general plan of urging patients, either on parole or after discharge, to continue to receive treatment at the hospital or at a proper clinic, and supplement this by a follow-up system for recording the results of such treatment. Furthermore, it is suggested that all patients with neurosyphilis be paroled for an indefinite period so that they may be continually supervised throughout the whole course of the disease.

In order to give the patients an opportunity to obtain the greatest amount of help and relief, it seems essential that all possible speed should be used in diagnosing cerebrospinal syphilis, and that all patients with suitable cases should receive an initial intensive course of arsenic and spinal drainage and mercury and iodids as soon as practicable after admission, in order to produce a cure if possible, or, failing this, to obtain the greatest amount of improvement.

If patients do not do well on large doses, it seems feasible to give smaller doses, with the idea of using neo-arsphenamin both for a certain amount of spirocheticidal effect and for a tonic action; in other words, to give the patient the benefit of the latest and best treatment whether this be from the direct method of specific treatment or from the standpoint of increasing the patient's resistance to the disease by the indirect method of building up the general bodily health and well-being of the patient.

THE OBJECTIVE FINDINGS IN THE PSYCHOSES *

PHILIP J. TRENTZSCH, M.D.

Senior Assistant Physician, St. Elizabeths Hospital

WASHINGTON, D. C.

The object of this paper is to bring forward an objective diagnostic symptom occurring in some of the psychoses. For many years, the need of objective symptoms as diagnostic aids has been felt, but so far few of these have been made available. Most of the symptoms now used in diagnosis of the psychoses are largely subjective, and if we look no further, our psychiatric knowledge must remain restricted. The outlook for the determination of objective signs in the psychoses becomes hopeful if one proceeds from the point of view of White,[1] which emphasizes the importance of "considering the individual as an organism of the whole." This necessitates the study of the patient from all possible directions.

The subject to which I wish to draw attention in this contribution concerns the relation of the neurocirculatory system to the psychoses. Considerable work has already been done with both the pulse rate and the blood pressure in psychotic persons, but up to the present time these factors have not been combined so as to determine a cardiovascular rating system that might have a diagnostic value.

During the world war, a system of rating was worked out by Schneider[2] by which the physical efficiency of men in the air service might be determined. It happened in the army service that some of the aviators gradually succumbed to physical and nervous deterioration. They became less reliable in handling their planes, and this led to accidents. For fear of being called yellow, the aviator would not tell when he did not feel in a condition to fly. It thus became necessary to develop some method by which his true condition could be determined.

With this in view, consideration was first given to the pulse rate in both recumbent and standing positions. Interesting data concerning pulse rates have been given by Cook and Pembrey[3] and by Meylan,[4]

---

* Read before the District of Columbia Medical Society, March 12, 1924.

1. White, William A.: Foundations of Psychiatry, Nerv. & Ment. Dis. Monograph Ser. No. 32, New York, Nervous and Mental Disease Publishing Co., 1921.

2. Schneider, Edward C.: A Cardiovascular Rating as a Measure of Physical Fatigue and Efficiency, J. A. M. A. 74:1507-1510 (May 29) 1920.

3. Cook, F., and Pembrey, M. S.: Observations on the Effects of Muscular Exercise on Man, J.Physiol. 45:429-446, 1913.

4. Meylan, G. L.: Twenty Years of Progress in Tests of Efficiency, Am. Phys. Educ. Rev. 18:441-445, 1913.

who has described differences in the rates in horizontal and vertical positions. Vierordt [5] states that there is an average increase of from twelve to fourteen beats in the standing posture. Crampton [6] comments that in vigorous persons the heart rate may increase as much as forty-four beats per minute.

In respect to the influence of exercise on pulse rate, Bowen [7] has found that the rapidity of the pulse during exercise is chiefly determined by, first, the speed of movement; second, the resistance encountered; third, the condition of the person; fourth, the age. This factor has also been discussed by Hartwell and Tweedy.[8] The decline in pulse rate after exercise has been investigated by Black and Bowdler.[9] They concluded from a study of the effects of stepping on a chair five times in fifteen seconds, that the heart rate in a healthy subject should not increase more than twenty-five beats and should return to normal within thirty seconds. Meakins and Gunson [10] have reported that after a climb of twenty-seven steps at a brisk pace, the pulse returns to normal within one minute in healthy subjects, while in patients to reach a normal required as much as five minutes.

Another factor that has been considered is that of the blood pressure in both standing and recumbent positions. Systolic pressures, according to Meylan,[4] are to be regarded as favorable signs when they range between 110 and 140 mm. of mercury for the horizontal position and 110 mm. to 150 mm. for the vertical. It has been stated by Dearborn [11] that adequate physical training raises the blood pressure.

That arterial pressure is altered by changes of posture, is commonly appreciated. Normally when one changes from the reclining to the standing position, the splanchnic vasomotor tone overcompensates for the hydrostatic effects of gravity. In normal persons, the systolic blood pressure is about 10 mm. higher in the erect than in the recumbent position. Erlanger and Hooker [12] found that change to the standing

5. Vierordt: Anatomische, physiologische und physikalische Daten und Tabellen, Jena, G. Fischer, 1908, p. 235.

6. Crampton, C. W.: Blood Ptosis Test, Proc. Soc. Exper. Biol. & Med. **12**:119-122, 1915.

7. Bowen: Am. Phys. Educ. Rev. **8**:232, 1903.

8. Hartwell, G., and Tweedy, N.: Some Effects of Muscular Exercise on Women, J. Physiol. **46**:9-11, 1913.

9. Black and Bowdler: Reports on the Air Medical Investigations Committee, London, No. 2, 1918, p. 12.

10. Meakins, J. C., and Gunson, E. B.: Pulse Rate After Simple Test Exercise in Cases of "Irritable Heart," Special Rep. M. Res. Committee, London, 1918, No. 8, p. 27; Heart **6**:284, 1917.

11. Dearborn, G. V.: Blood Pressure in the Leg, Am. Phys. Educ. Rev. **20**:337-352, 414-423, 1915.

12. Erlanger, J., and Hooker, D. R.: Experimental Study of Blood Pressure and Pulse Pressure in Man, Johns Hopkins Hosp. Rep. **12**:145-378, 1904.

position might produce either a slight rise or fall in the brachial systolic pressure. Interesting data on changes in the systolic pressure have also been given by Hill [13] and Sewall.[14]

From the six sets of observations, a system of rating was worked out as follows: Of a group of fifty-four cases, Schneider [2] found that only six had a score ranging between 9 and 1. This showed that 88.8 per cent. had a low score. These persons were then examined independently by the neurologists and internists without knowing the data regarding their cardiovascular ratings. These examinations revealed that abnormal conditions were present in thirty of the forty-six cases. "Thus, when working independently 62.5 per cent. of the group of forty-six with low scores by the cardiovascular efficiency tests were found by others to be below standard."

Lewis,[15] in a recent monograph, has contributed data which are of interest in this connection. In this, he reports that in a study of 601 cases of dementia praecox occurring in a series of 4,800 necropsies in cases of mental disease, observations were recorded as to age, color, sex, lethal lesions and the general condition of the circulatory system, all of which were correlated with the weight of the heart. He included among the dementia praecox cases only those of the hebephrenic and catatonic type or combinations of these. His investigations had convinced him that there were such fundamental differences in the constitutional make-up, in the morbid anatomy as well as the clinical features, between these and the paranoid types as to require their separation in this study. In general, Lewis found that there was a small circulatory system in cases of the dementia praecox group. This constitutional feature was independent of age, color, sex and duration of the psychosis or associated disorder. He expresses the opinion that it is not a question of an arrested development of the circulatory system, but rather that the system lacks ability to react by a satisfactory compensation hypertrophy when occasion demands, and often remains below average size even after developing valvular insufficiencies. While ordinarily the heart grows in size as age advances, this does not occur with the constitutionally small heart.

Among the 601 cases of dementia praecox, there were 430 hearts of less than average weight and 171 weighing more than the average.

13. Hill, L. B.: Influence of the Force of Gravity on the Circulation of the Blood, J. Physiol. **18**:15-53, 1895.

14. Sewall, H.: On the Clinical Significance of Postural Changes in the Blood Pressure and the Secondary Waves of Arterial Blood Pressure, Am. J. Med. Sc. **158**:786-816, 1919.

15. Lewis, Nolan D. C.: The Constitutional Factors in Dementia Praecox, with Particular Attention to the Circulatory System and to Some of the Endocrine Glands, Nerv. & Ment. Dis. Monograph Ser. No. 35.

In 71.55 per cent. of his cases, there was a small aplastic heart. In this series there were 204 patients with aplastic aortas, 136 in which the aorta was diseased, and 261 in which it was not abnormal. In a table giving comparative weights of the heart in the psychoses it is shown that 75.5 per cent. of the hearts in cases of dementia praecox (hebephrenic and catatonic) weighed less than the average, while of the paranoid cases only 8.7 per cent. were below the average. The heart was of less than average weight in 30 per cent. of manic-depressive cases,

TABLE 1.—*Individual Neurocirculatory Ratings in 124 Cases of Mental Disorders*

Number of Cases									
23	15	31	16	9	8	7	8	2	5
Dementia Praecox Paranoid	Dementia Praecox Catatonia	Dementia Praecox Hebephrenia	Dementia Praecox Mixed	Manic Depressive	Epileptic	Psychosis with Mental Deficiency	General Paralysis	Mental Defective	Psychosis with Psychopathic Personality
10	12	6	14	15	8	13	9	9	5
15	2	2	18	15	9	7	12	14	16
13	7	2	11	11	10	9	10	..	12
3	10	9	10	16	8	12	10	..	12
15	9	6	12	12	1	11	4	..	11
2	11	7	6	3	10	6	7	..	..
17	2	0	13	11	1	12	15	..	..
10	0	8	12	15	5	..	7	..	..
12	7	5	1	15	..	..	..	..	..
14	1	6	9	..	..	..	..	..	..
14	10	9	12	..	..	..	..	..	..
12	9	14	14	..	..	..	..	..	..
13	3	9	6	..	..	..	..	..	..
7	3	2	12	..	..	..	..	..	..
15	4	10	16	..	..	..	..	..	..
13	..	2	17	..	..	..	..	..	..
12	..	6	..	..	..	..	..	..	..
11	..	3	..	..	..	..	..	..	..
8	..	1	..	..	..	..	..	..	..
11	..	3	..	..	..	..	..	..	..
2	..	14	..	..	..	..	..	..	..
5	..	9	..	..	..	..	..	..	..
8	..	5	..	..	..	..	..	..	..
..	.	13	..	..	..	..	..	..	..
..	..	9	..	..	..	..	..	..	..
..	..	5	..	..	..	..	..	..	..
..	..	4	..	..	..	..	..	..	..
..	..	3	..	..	..	..	..	..	..
..	..	9	..	..	..	..	..	..	..
..	..	6	..	..	..	..	..	..	..
..	.	8	..	..	..	..	..	..	..

30.2 per cent. of epileptic; 59.1 per cent. of paretic; 22.2 per cent. of cases of senile dementia; 21.3 per cent. of arteriosclerosis and 37.2 per cent. of cases of mesoblastic cerebral syphilis. Such observations show a striking contrast between the weight of the heart in the catatonic and hebephrenic types of dementia praecox and other forms of psychoses, particularly those of the paranoid and manic-depressive groups.

The occurrence of cyanosis in cases of mental disorder has been studied by Cornell.[16] In 241 cases, he found that 78 per cent. showed

---

16. Cornell, W. B.: Cyanosis in Dementia Praecox, J. A. M. A. **59**:2208-2210, 1912.

cyanosis of the extremities, varying from a mild degree to an extreme dusky purple. This was often noticeable elsewhere in the skin, particularly over the upper part of the back, face and lips. It was an inconstant phenomena and varied from day to day. He observed that cyanosis occurred in 90 per cent. of cases of catatonia, 75 per cent. of hebephrenic and 50 per cent. of those of the paranoid type. It was present in only two of thirty-six cases of manic-depressive psychosis.

From the foregoing observations, it has been suggested that it might be possible to formulate a neurocirculatory rating that might have an application in the study of mental disorders. With this idea in mind, I have carried through a series of observations in 124 cases of mental disorders of various types. The cases studied were those of white men ranging between 20 and 50 years of age. The diagnoses in most of the cases were determined in conferences of the hospital staff. All of these

TABLE 2.—*Percentage Ratings in Mental Disorders*

	Number of Cases	Number of Cases Rating 10 Points or More	Number of Cases Rating under 10 Points	Percentage Rating of More than 10 Points
Dementia praecox paranoid.................	23	16	7	73.1
Dementia praecox catatonic.............. ..	15	4	11	26.6
Dementia praecox hebephrenic..............	31	4	27	12.8
Dementia praecox mixed type..............	16	12	4	75.0
Manic depressive psychosis.................	9	8	1	88.8
Psychosis associated with mental deficiency.	7	4	3	56.8
Epileptics.....................................	8	2	6	25.0
General paralysis.............................	8	4	4	50.0
Psychosis with psychopathic personality....	5	4	1	80.0
Mental defectives..............	2	1	1	50.0
Total....................................	124			

patients underwent a series of neurocirculatory tests. The ratings obtained in each case are given in Table 1.

This chart shows contrasts among the ratings in different types of psychoses. High ratings occur in greater frequency in paranoid and manic-depressive psychoses, while in catatonia, hebephrenia and epilepsy they are generally low. These results have certain correlations with the observations of Lewis in his study of the necropsy findings in the circulatory system among the psychotic. The small aplastic hearts that he found in cases of catatonia and hebephrenia could not be expected to give as high a rating as those with a normal circulatory system. One might expect a similar relation to exist among epileptic patients, who also show low ratings.

In Table 2, the percentage relations of the ratings are more clearly shown.

It is here shown that sixteen of every twenty-three patients with paranoid dementia praecox and eight of nine with manic-depressive

psychosis had high ratings, while in the hebephrenic form of dementia praecox, only four out of thirty-one, and in the catatonic form, four out of fifteen, had high ratings.

Circulatory relations in manic-depressive psychoses have been studied by a number of investigators. Rehm [17] has observed that there is in this disorder a very high pulse rate, not only in phases of excitement, but also in those of depression. He suggests that this may be a point of some value in the differential diagnosis between manic-depressive psychoses and dementia praecox. Stransky [18] found in melancholia that the blood pressure was changeable, but was usually high. In manic-depressive psychoses, the blood pressure was less, becoming high only when the patient became angered. Enebuske [19] concludes that blood pressure has a diagnostic value. Using the sphygmomanometer, he found that there existed a vasomotor restlessness in manic-depressive psychoses when there was a condition of hypertension, and that this was limited in its duration. In dementia praecox this was observed in every condition. Pfortner [20] found that there was sometimes a slight arrhythmia of the pulse in dementia praecox. · The pulse variations that are so characteristic for functional psychoses were almost always absent. The blood pressure was usually low, about 93. It seldom was above 130, and the highest observed was 152. Weber [21] found that the systolic and diastolic pressures were higher in manic-depressive psychoses than in dementia praecox. Similar results have been noted by Cramer and Craig.

In order to find some explanation for the occurrence of low ratings in some of the cases of the paranoid forms of dementia praecox and the high ratings in some of the hebephrenic and catatonic cases, a study was made of twenty-five specially selected cases. The patients selected were placed under the direction of the physical director of the American Red Cross. After having been weighed and measured, they were given a standard series of exercises for one month. At the end of this period, they were again weighed and measured, and a new rating was made. The results of this experiment are given in Table 3.

The data in Table 3 show that in the paranoid cases there was a definite response to the exercise. All the patients showed slight increase in muscular development and one, V. D., who rated two points before the exercises, went up to thirteen points at the end of the test. No response was shown by the catatonic and hebephrenic patients. Patients with epilepsy also showed no improvement. They were inert and unable

17. Rehm, O.: Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:263, 1922.
18. Stransky, E.: Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:263, 1922.
19. Enebuske: Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:264, 1922.
20. Pfortner, O.: Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:263, 1922.
21. Weber: Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:264, 1922.

to hold their first rating. In the manic-depressive cases, the patient who lost was passing into a depressed state before his series of exercises was completed. Whether or not this explains the difference in rating, I am not prepared to state.

At this point the question was suggested as to whether the glands of internal secretion might have some bearing on these cases. Stuurman [22] has found that subcutaneous injection of 0.7 mg. of epinephrin will in certain cases produce an increase in the systolic blood pressure and pulse rate. The diastolic pressure in a few cases was found to be increased, but usually it fell or remained unaltered. In schizophrenia, the reaction was less marked than in other psychoses. The reaction was more intense in the paranoid forms than in the catatonic or hebe-

TABLE 3.—*Ratings Before and After Taking Exercise*

	Before	After
Dementia praecox, paranoid........................	15	11
	10	11
	13	17
	13	15
	2	13
Dementia praecox, catatonic.......................	0	0
	7	7
	9	7
	12	9
Dementia praecox, hebephrenic....................	6	6
	2	7
	9	9
	12	18
Dementia praecox, mixed..........................	14	6
	3	1
Manic-depressive.................................	11	9
	15	7
Epileptic...	9	8
	8	6
Psychosis with mental deficiency..................	13	15
General paralysis.................................	12	7
	10	15
Mental defective..................................	2	9
Psychosis with psychopathic personality..........	12	16

phrenic. Patients with epilepsy showed a diminished reaction in pulse and blood pressure. In manic-depressive psychoses, there was a rather intense reaction, especially in those that were restless. Goetsch [23] has found that the injection of 0.5 c.c. of a 1 : 1,000 solution of epinephrin chlorid given after a period of ten minutes causes a rise of about 10 points in the pulse or systolic pressure, or in both, and that this is associated with definite subjective and objective symptoms. "This," he comments, "is an indication of hypersensitiveness of the sympathetic nervous system. . . . On the other hand I am not prepared to say that all cases which show a positive reaction are, therefore, hyperthyroidism. . . . However, we do know that the test is positive in all cases of hyperthyroidism, a fact which is founded upon abundant

22. Stuurman, F. J.: Nederl. Maandschr. v. Genees. (Leiden), **3**:44-60, 1922.
23. Goetsch, E.: Hypersensitiveness Test, Endocrinology **4**:389-402, 1920.

physiological research carried out particularly by Cannon and Levy."
Pedro Escudero [24] has stated "We have already proved in contradistinction to Goetsch's statement, that the reaction produced by the injection of 0.5 mg. of adrenalin is not specifically due to a state of hyperthyroidism, that the hyperfunction of the thyroid gland can exaggerate the action of adrenalin. . . . We consider an accelerated heart after its administration a sign of sympathicotonia. . . . Tachycardia is provoked; we consider this a positive test. . . . Acceleration of fifteen to thirty is considered tachycardia; ten to twelve is a weak positive."

The twenty-five patients that had been put through the exercise experiment were given epinephrin tests, and records taken every two

TABLE 4.—*Results of Fluoroscopic Examination in Ninety-Nine Subjects*

Types of Cases	No. of Cases	Tubular	Normal
Dementia praecox, paranoid type	21	8	13
Dementia praecox, catatonic	15	10	5
Dementia praecox, hebephrenic	31	24	7
Manic-depressive	6	5	1
Epileptic	9	8	1
Psychosis with mental deficiency	10	3	7
General paralysis	7	2	5
Psychosis with psychopathic personality	1	0	1

TABLE 5.—*Roentgenographic Findings*

Types of Cases	No. of Cases	Normal	Tubular
Dementia praecox, paranoid type	20	12	8
Dementia praecox, catatonic	23	5	18
Dementia praecox, hebephrenic	11	2	9
Manic depressive	4	4	0
Epileptic	6	0	6
General paralysis	5	5	0
Psychosis associated with mental deficiency	9	2	7
Psychosis associated with psychopathic personality	2	1	1

minutes for a period of ten minutes. Three of the patients showed a rise of 10 points or more in the pulse rate, or blood pressure or both. These three were given a complete Goetsch test, but not one of them showed a reaction that could be interpreted as typically positive. These findings should eliminate twenty-two as not having circulatory disturbances primarily due to endocrinologic factors. The three that showed atypical findings could also be eliminated.

Following the completion of these tests, a group of ninety-nine subjects were given a fluoroscopic examination by the roentgenologist to determine the form of the heart. The results of these observations are given in Table 4.

24. Escudero, P.: Pilocarpine and Atropine Tests, Endocrinology 7:305-310, 1923.

These findings cannot be regarded as accurate as it was necessary to make the comparative measurements in a few seconds. It is, however, of interest to note how these various observations correlate with the former experiments.

In the group of the paranoid forms of dementia praecox, the majority of the patients had a normal heart, while in the hebephrenic and catatonic group the majority had a heart of tubular form. This particular type of heart was also in the majority in the epileptic and manic-depressive groups. Such results do not harmonize with other ratings obtained for these groups. In order to confirm these findings, a roentgenogram was made of each heart. We used as a guide in the reading of the measurements that were made that given in the U. S.

TABLE 6.—*Summary of Data Obtained from Various Experiments in Twenty-Five Cases*

Types of Cases	Neuro-circulatory Index		Neuro-circulatory Index after Exercise		Endo-crinology		Fluoroscopic Findings		Roentgeno-graphic Findings	
	High	Low	High	Low	High	Low	Tubu-lar	Nor-mal	Tubu-lar	Nor-mal
Paranoid dementia praecox..............	5	1	6	0	1	5	3	2	1	3
Hebephrenic dementia praecox..............	1	3	14	4	1	3	3	1	3	2
Catatonic dementia praecox..............	1	3	0	4	1	3	2	..	2	..
Mixed type dementia praecox..............	1	1	0	2	0	2	..	..	..	..
Manic-depressive.......	2	0	1	1	0	2	..	1	..	1
Epileptic...............	0	2	0	2	0	2	1	1	2	0
Psychosis associated with mental deficiency................	1	0	1	0	0	1	2	..	1	..
General paralysis......	2	0	1	1	0	2	..	..	..	..
Mental defective.......	0	1	0	1	0	1	..	..	..	..
Psychosis associated with psychopathic personality..........	1	0	1	0	0	1	..	..	..	..

Army X-ray Manual.[25] Among twenty persons with the paranoid form of dementia praecox, there were twelve that had a normal heart and eight in whom the heart was of tubular form. Among twenty-three patients with the hebephrenic form, there were eighteen in whom the heart was tubular and five in whom it was normal. Among eleven patients with catatonia, there were nine with tubular heart and two that were normal. The observations on the hearts of those with manic-depressive psychosis were quite different in the photographs from the fluoroscopic examinations. In four cases, the heart was normal in form. These photographic observations are recorded in Table 5.

25. U. S. Army X-Ray Manual: X-Ray Examination of the Heart, pp. 402-403; Method of Heart Measurement, pp. 412-418, New York, Paul B. Hoeber, 1920.

The results obtained in all of these various experiments tend to confirm the findings of the first experiment.

A summary of all the data obtained from the various experiments of the twenty-five cases is given in Table 6.

SUMMARY

1. In 601 necropsies in cases of the hebephrenic and catatonic types of dementia praecox, 71.55 per cent. were found to have small aplastic hearts. The incomplete development involved also the capillary system.

2. In a study of the weight of the heart in the psychoses, 75.5 per cent. of the patients with dementia praecox had hearts of less than average weight, while only 7.8 per cent. of the patients with paranoia had hearts of less than average weight; 30 per cent. of the patients with manic-depressive psychosis and 30.2 per cent. with epilepsy had hearts weighing less than the average.

3. A neurocirculatory rating system was worked out for aviators. It was found that the men with a low rating did not do well in their flying. Those with a high rate seemed to be rather stable types.

4. A group of patients suffering from psychoses were given various tests to determine a neuropsychiatric rating. A high rating was obtained in only 18.8 per cent. of cases of the hebephrenic type of dementia praecox and in 26.6 per cent. of those of the catatonic type. In the two groups, 82.61 per cent. of the cases had a low rating. In comparison with this, Lewis found 71.55 per cent. of small aplastic hearts in the same types of cases. In the paranoid group, the neurocirculatory rating was high in 73.1 per cent. In this group, Lewis found only 8.7 per cent. of hearts weighing less than the average. The neurocirculatory ratings obtained in manic-depressive and epileptic cases compare favorably with the necropsy findings of Lewis in these disorders.

5. After a series of exercises, the ratings of paranoid patients increased, while the hebephrenic and catatonic cases did not respond.

6. A group of patients were tested for endocrinologic abnormalities. Only three of these showed any indications of a glandular disturbance. This would seem to indicate that the glands of internal secretion had little or no bearing on the pulse rate or blood pressure of the remaining cases.

7. The fluoroscopic and roentgenographic findings compared favorably with the other ratings.

In conclusion, we would state that if the foregoing findings are correct, it would seem that in the neurocirculatory rating one has a definite objective symptom of the psychoses, and this suggests a physical reason for mental illness as well as those that are psychic. It is not advisable to ignore either one.

PARTS OF CENTRAL NERVOUS SYSTEM WHICH TEND TO EXHIBIT MORBID RECESSIVE OR DOMINANT CHARACTERS *

SMITH ELY JELLIFFE, M.D., Ph.D.

NEW YORK

In the present state of our knowledge relative to clinico-anatomo-pathologic correlations with ontogenetic and phylogenetic problems, the hereditary mode of investigation offers much for a more comprehensive understanding of structure and function of the nervous system. There are promising opportunities for productive research even though the subject discussed is yet in a stage in which it defies precise formulation.

The human body, like all living matter, is an energy system for the capture, transformation and release of energy. Specific receptors have been evolved for the capture; definite organs have become structuralized to handle the incoming energy and transform it, so that effector organs of varying complexities may discharge it.

The human body is not a closed system, and any adequate understanding as to what happens and has happened must include the outside as well as the inside relationships. In a way, inside, we see structure; in a sense, function may be said to come initially from outside. Experience, from the beginning, has engraved itself into living matter. Stimulus, outside, has cut into matter, inside, and through engram binding has built up that mnemic (instinct) capacity to reproduce reaction to stimulus, which structurally we call an organ, and physiologically we call its function. An organ, then, is a bit of structuralized experience of enormous mnemic capacity to handle in a definite and reliable manner this interchange of energy inflow and behavior outgo.

We would here attempt to envisage the phyletic story of the laying down of structures, and what is even more subtle, to visualize the evolution within structure of increasing functional complexity. One might say that over a copper telephone wire there may travel simple sound waves, more complex dots and dashes, still more highly evolved grunts and gurgles, laughter and sobbing, then rich speech of many languages, then highly complex mathematical formulations such as an animated discussion of calculus or conic sections or noneuclidian geometry. What childish intelligence is it that would think that by looking at a cross section of the wire one could see all these things streaming through it? Or, as there are successive developments of a lithograph

---

pattern from its initial outline through its dozen or more overlays until the final pattern emerges, so does there develop within organs, themselves changing to meet the new influx, the functional patterns of evolutionary engram accumulations (Semon).

No organ, seen structurally, alone can ever reveal the secrets of its functional complexities which a billion years of engram inheritance has given it. Notwithstanding this insuperable difficulty, can it be formulated how far the parts of the nervous system show some definite form of inheritance which evidences itself in manifest disturbances of function to which we can provisionally attach some symbol, i. e., name as a disease?

It is clearly to be seen from the simplistic telephone wire or lithograph imprint analogies that for the present the most fruitful mode of approach may be through the building-up process which we call inheritance by the engram pattern route. *Parts of the nervous system shall be taken, as far as possible, to mean functional units; organs handling experience in as primitive a manner as can be seen behind the complexities: mnemic and instinct patterns, i. e., neurons and neuron systems.*

THE ENERGY FLOW

It is first imperative to scrutinize the incoming and the outgoing energy flow in order to get any insight into the inner functional secrets of the transformer. It would seem that a logical partition should be set up between the receptive and the emissive side of the organism's patterns, since the channeling of the common discharge pathways has resolved the emergent patterns into few structural effector types, whereas the incoming streams are caught by multiform devices. Where in the mechanisms between the extended receptor intake and the limited effector outflow, as seen in functional purpose, may we be able to distinguish such modified structuralizations of function caught at inferior, i. e., more primitive, stages of engram capacity, which a former group of students of the diseases of the nervous system termed *atavisms,* and which today we are discussing under the conception of *heredodegenerations?* These heredodegenerations, as recorded in clinical experience, may be considered rare, yet they are frequent enough and of sufficient importance to discuss.

As a preliminary, the discussion of certain aspects of a differentiation of hereditary (biologically considered) from developmental factors is imperative.

Whereas the newer researches of a host of students of genetics, to mention only Morgan, Bridges, Correns, Sturtevant, show that much more precise criteria are available for distinguishing the differences between developmental and hereditary conditions, combinations of these factors still remain constantly operative in human pathology. It must

be held that the exclusive transmitting mechanisms, the genes, are located in the chromatin, but developmental, or environmental factors of a great variety of kinds may not permit the expression of the developing factors. To complicate still further this already overdetermined situation, Plate with others would also posit the possibility of inhibiting factors in the genes themselves. These tend to hinder the development of an organ or a part of the organ.

HEREDITARY FACTORS

As Stockard [1] puts it, a child may inherit the exact nose pattern of its father, yet it may for some cause entirely fail to develop a nose. The inheritance was perfect, but the ability to express the structure was lacking.

The human organism is so closely integrated that no matter where the defect appears, either in its genetic or developmental accent, all other parts of the body are also implicated. One turns, as in all analysis, first to extreme states; let us follow Stockard's discussion of this aspect. Fortunately, his first chosen example lies well within the neuropsychiatric field: cretinism—a child born with little or no thyroid gland. Is the absence of the gland, or of its activity, hereditary, or is it developmental, or may it be both? The cretin is a thyroid dwarf. Without thyroid stuff it cannot go further. It remains—varyingly of course—in an early larval condition of man. Without thyroid, he will not metamorphose into an adult. But the cretin, for our purpose, is not a *type*. He expresses an extreme growth condition—a pathologic state; certain dwarfs, however, constitute types, i. e., definite genetic situations. Many are normal, differing only as some small breeds of domestic animals may differ from larger ones; the African pygmy is first chosen. In some respects, they resemble cretins. Are they genetic types or have they grown in an unfavorable environment?

True hereditary factors, Stockard reminds us, may be found in another type of dwarf—the achondroplastic dwarf. Whereas both hypophysis and thyroid, here viewed as parts of the endocrinovegetative nervous system, have been held responsible—as developmental factors —both the human achondroplastic and his numerous canine homologues, French and British bull dogs, dachshund, Pekingese, etc.—seem to show, viewed from the genetic point of view, that the achondroplastic complex may be a mendelian dominant that is viable only in the heterozygous form. When there is a double dose of the dominant and the zygote is homozygous, lethal expression follows, and the child is incapable of living after birth. (Are albinos similarly analyzable?) Morgan has pointed to a number of instances of this homozygous lethal dominant.

1. Stockard: Harvey Lectures XVII, 1923.

Thus, a dominant character (or structure, new species—genius, etc.) may be unable to establish itself in a homozygous condition and may never become abundant in the race. Stockard here makes an interesting observation that whereas the double dominant might give a lethal effect in man, it may give rise to a pure homozygous breed in lower animals.

ACHONDROPLASIA

The general effort to explain achondroplasia as being due solely to developmental (or genetic) glandular insufficiencies, needs some revision when one considers partial or localized achondroplasias (see other abiotrophies, in Growers' sense, localized hemifacial atrophies, localized hemitrophic or dystrophic edemas; various muscular atrophies or dystrophies). If the internal secretions in the blood (I believe that a too strict humoralism relative to the endocrins is a limited concept) produce these peculiar growths, why do they not always act in all similarly growing parts in a similar manner? Numerous discrepancies can be pointed out in micromelic humans, dachshund and basset dog, who have short legs and hound heads. To avoid the difficulties, one could assume an inherited type of gland defect, and an inherited susceptibility of a humerus or a femur, or part of the nervous system peculiarly sensitive to the hormone stimulus, which after all is just a collection of words.

Midgets (ateliosis) constitute another genetic type of mixed characters. As the opposite extreme are giants—acromegaly, general or localized. Here again certain breeding experiments in dogs throw some light on this subtle group of interrelated developmental and genetic factors. Hypophyseal factors are well recognized. In dogs, the St. Bernard and mastiff types show definite acromegalic features, as well as being giant dogs. Here the glandular structure is hereditarily chosen.

Jendrassik,[2] has said that observation has shown that nearly all the tissues of the body can suffer some form of hereditary degeneration. Not all of the tissues of an organ may be involved, but special ones. Thus, in the nervous system single systems may show the degeneration situation—muscle groups among others. These may show great variability, hence the unending kaleidoscopic clinical pictures, with their consequent labeling as different diseases, out of the multiplicity of which Jendrassik sought to establish some uniformity. There were familial forms of hereditary spastic paralysis, disturbances of vision, optic atrophies, eye muscle palsies, nystagmus, speech disturbances, athetoses, tremors, ataxias, difficulties in swallowing, and various degrees of mental enfeeblement. There were also transition stages between Marie's

---

2. Jendrassik, in Lewandowsky: Handbuch der Neurologie, Berlin, Springer **2**:321, 1911.

heredocerebellar ataxias and Friedreich's ataxias, and various mixed forms of the same.

<center>HEREDITARY GROUPINGS</center>

All of these disease trends, whose common basis is some characteristic type of degeneration, show a more or less identical course in the affected families, and yet the shifting mosiac may be understood in terms of variations in involvement of the various elements making up the organs. Thus there arise pathologic alterations in the nervous system, the muscles, the tendons, the bones, the vessels, the receptor organs and even the metabolism. Out of this medley, Jendrassik would construct different groups. Following largely Déjerine's older and now superseded separation principles, he would gather these behind etiologic concepts, such as: (1) forms with latent hereditary; (2) consanguinity forms: spastic paralysis, retinitis pigmentosa, deaf and dumbness, etc.; (3) double heredity (recessive latent) albinism, and (4) sexual differentiation forms—in which the females of the involved families remain sound and the males are involved. The direct descendants of the males remain sound; the male descendants of the sound females are involved. Hemeralopia, night blindness, optic atrophy, some dystrophies (?) and Merzbacher's aplastica extracorticalis occur.

The difficulties of such grouping conceptions, which Siemens[3] remarks belong to the "Rumpelkammer," apart from their insufficiencies from present day genetic concepts (akin to our own difficulties), Jendrassik thought more or less insuperable, and leaves them for what he considers a practical formulation on a clinical basis. Going deep down in the biologic foundations, he attempts to formulate two principles: chemical and physical, which show interacting trends. As chemical hereditary degenerations, he would review all hereditary endogenic metabolic disturbances, such as obesity, thinness, gout, diabetes, hemophilias, cystinurias, alkaptonurias, albinism and certain immunity situations of complex and purely fictional logical value, through which portal he would show a descent to the older Hippocratic suspicion that all disease shows hereditary possibilities. Into this morass a pessimistic logic as to the real insolubility of all problems having so many millenia of phyletic development would lead us all. Heredity thus comes really to mean nothing but "it happens."

In Jendrassik's physical group, many forms are assimilable. For by "physical," he means "structural" involvements. Jendrassik tries to enter into the mysteries of the phyletic developmental backgrounds of the different parts, not of the nervous system, for he suspects that it is only a coordinator of the organism as a whole, but at first into the

---

3. Siemens, H. W.: Einführung in die allgemeine und spezielle Vererbungspathologie des Menschen, Ed. 2, Berlin, Springer, 1923 (full bibliography).

muscular anomalies, seen as hereditarily involved. Here he stands on the doubtful assumption that the muscles are *primarily* implicated. Muscle and nerve, at bottom, are one, as Parker and others have shown. This dictum, however, does not prevent a pragmatic dealing with them as separate systems. In this connection, it can be understood why degeneration of the long corticospinal fiber tracts may come under microscopic or even macroscopic review. Whether Bing's [4] notion of a cortical aplasia is the valid interpretation of these defective developments, or the cortical aplasias are resultants of defective gravimetric and inertia handling from the receptor-muscular adjustments, is an open question. Here may be seen the essential error of the orthodox conception, already pointed out, that the human energy system is a closed system. Motor nuclei, whether cortical, medullary or spinal, conceived of as being deficient in energy and hence leading to the abiotrophies, is a faulty concept in the scheme I primarily advocate.

Jendrassik leans heavily on the generalization founded on the degenerations seen in that outstanding familial hereditary anomaly known as amaurotic idiocy. Here the results of the defective processes in development appear early (clinically) and are matched by the early defects seen in the small retinal nerve cells, the smaller spinal cord (even macroscopically demonstrable). Similar cord defects are not isolated (Friedreich, Bing, Kollarits).

Londe would group these in some unified form, but, following a purely metameric scheme, he leaves us in the ditch as to the essential structural defect. Nevertheless, his differentiations may be utilized from their negative side: heredodegenerations at the cerebellar level (cerebellar heredo-ataxias), brain stem level (familial bulbar palsies, familial ptoses), spinal cord level (Friedreich, spastic paraplegias), brain plus spinal cord (familial diplegias) and various mixtures. Bing and Higier [5] attempt to clear up these interwoven intricacies, but leave us with watery foundations from which Jendrassik essays to deliver us by positing focalization of striking characteristics—thus, dystrophic spastic paraplegia, Friedreich types (the purely artificial nature of which he acknowledges) and other then available material (1911), leaving one in the lurch as to a possible philosophical coordination. Crouzon does not go further.

When Jendrassik speaks of a worthy principle in the "course" of these many kaleidoscopic variations, we cannot follow him. He strains

---

4. Bing, R.: Köngenitale, heredofamiliäre und neuromuskuläre Erkrankungen, in Mohr and Staehelin: Handbuch der Innere Medizin, Berlin, Springer 5:1912.

5. Higier: Zur Pathologie der angeborenen familiären und hereditären Krankheiten Spezielle der Nerven und Geisteskrankheiten, Arch. f. Psychiat. 48:41, 1911.

at a monstrous artefact which has no value for our study. Similarly his "Diagnostik." Notwithstanding this effort, something may be gained in utilizing his printed words as of purely fictional (i. e., in their logical function) value. In Gardner's case, a mother showed nystagmus and intention tremor, the oldest daughter had in addition to these signs also a spastic paraplegia, with increased tendon reflexes, while three younger sisters had lost tendon reflexes; the youngest was scoliotic. He also cites a case of Higier with a mixed syndrome. Two siblings had hereditary optic atrophy, a third showed cerebellar ataxia, a fourth amaurotic idiocy. *Something* of primary character lay behind these. Was it a system factor, interpretable in terms of a "part" of the nervous system following a mendelian generalization? Jendrassik envisages the problem in the aspect of a "heteromorphous heredity," the more precise definition of which we would attempt if possible. Complicating temporal evolution factors are seen by him. Thus the increased and lost tendon reflexes are envisaged as purely temporal stages in the evolution of the syndrome.

Jendrassik's general groups are as follows: (1) dystrophies, (2) spastic heredodegeneration, (3) ataxic heredodegeneration, (4) intermediary forms. Bing's classification is similar but even more symptomatic, and hence really less available for strict hereditary analysis.

Jendrassik's clinical groups include: (1) muscular dystrophies, with their subordinate forms: (*a*) pseudohypertrophic, (*b*) Erb's juvenile form, (*c*) Leyden's types, (*d*) Charcot-Duchenne types, (*e*) Zimmerlin types, (*f*) hereditary dystrophies of Jendrassik, (*g*) pseudocontractures and pseudo-anchyloses; (2) dystrophies with attendant peripheral nerve degenerations: (*a*) Charcot-Marie (Neural-Hoffmann [6] type), peroneal-tooth type—Eichhorsts' family tree being of special significance, also others (?), (*b*) Werdnig-Hoffmann types, (*c*) bulbar types, (*d*) Gombault-Mallet (Déjerine-Sottas) types, (*e*) Eichhorst types, (*f*) mixed and aberrant forms. Jendrassik also describes hereditary ptoses and other muscle palsies, bony dystrophies, acromegaly and giantism, dwarfism, and other types. These involve other "parts" of the nervous system, especially the "pituitary" part, for their placing in the general scheme of things.

Jendrassik then passes on to the hereditary structural involvements of the spinal cord (a conglomerate and not a unitary system, as we understand "parts" of the nervous system). Here clinical terms (spastic paraplegia) mar the picture, and Jendrassik clearly shows that clinical neurology and pathologic uniformities are badly unified. Although the broad term of infantile cerebral palsies (Freud) was involved, the "parts" of the nervous system involved are so diffuse and discordant

---

6. Hoffmann, H.: Vererbung und Seelenleben, Berlin, Springer, 1923.

as to negative any possibility of a coordination in the sense sought by us. A vast variety of exogenous and endogenous factors are implicated which for the present absolutely prohibit any philosophic unification, in spite of the fact that Jendrassik would essay to deal with them as a unicum.

Jendrassik, then, beyond giving us a panorama of crude clinical forms, only affords a general material assortment to which further material from scores of observers may be added. Biologic hereditary conceptions are entirely absent, and his material offers no help along these lines.

BIELSCHOWSKY'S GROUPING

This leads us to the formulations arrived at by Bielschowsky[7] as tentative schemes for further study. Bing's principles lean too heavily on isolated symptoms; Jendrassik's clinical groupings further the analysis but lack deeper significance. Schaffer's[8] emphasis on the histopathologic differentiations is an important contribution to the problem, and Bielschowsky's biologic formulation is the most advanced available.

Bielschowsky offers as a tentative grouping:

I. *Pure Dysplasias*: Malformations from Organic Disturbance.
   (*a*) Malformations of Pallium: mikropolygyria, pachygyria, agyria.
   (*b*) Malformations of Striatum: état marbré of Vogt-Marie.
   (*c*) Malformations of Caudal Parts of Central Nervous System: micromelia, syringomyelia (some forms).

II. *Dysplasias with Blastomeric Complications*: tumor formation, tuberous sclerosis.

III. *Abiotrophies*:
   (*a*) Abiotrophies with Blastomeric Complications: pseudosclerosis and diffuse sclerosis.
   (*b*) Abiotrophies with Local Total Necrosis of the Parenchyma:
       (1) Necrosis of the putamen and globus pallidus (Wilson's disease and dystonia lenticularis).
       (2) Necrosis of the globus pallidus (before this not well known—Fischer's case).
   (*c*) Abiotrophies with Elective Necrobiosis of Ganglion Cells:
       1. (*a*) Universal uniform cellular degenerations in all parts of the central nervous system, inclusive of the sensory receptors: amaurotic family idiocy, infantile form (Tay-Sachs).

---

7. Bielschowsky: Entwurf eines Systems der Heredodegenerationen, Jour. f. Psychiat. u. Neurol. **24**:48, 1919.

8. Schaffer, K.: Arch. f. Psychiat. **64**, 1922; Beiträge zur Lehre der zerebellären Heredodegeneration, J. f. Psychiat. u. Neurol. **27**:12, 1921; Tatsachliches und Hypothetisches aus der Histopathologie der infantil—amaurotischen Idiotie, Arch. f. Psychiat. **64**:21 and 570, 1920; Zur Pathologie und pathologischen Histologie d. spastischen Heredodegeneration, Ztschr. f. Neurol. **73**:101, 1922; Schweitz. Arch. f. Neurol. u. Psychiat. **7**:193, 1920.

      (b) Universal cellular degenerations with particular involvement of definite organ regions: juvenile amaurotic idiocy.

   2. Elective degeneration of cells of distinct organ regions; system disease in narrow sense.

      (a) Nucleus caudatus, and nucleus lentiformis: Huntington's chorea.

      (b) Cerebellar systems: cerebellar heredo-ataxias in many modifications.

      (c) Corticomotor systems: Spastic spinal paralysis, amyotrophic lateral sclerosis, spinal muscular atrophies.

These and related considerations too numerous to justify citation and too variable to be encompassed as yet within adequate generalizations, show that the situation can be glimpsed as a practical issue, and when clinico-anatomopathologic findings are coordinated and ranged in line with ontogenetic and phylogenetic correlations, we shall, as Charcot states, get a glimpse at some "diseases" due to definitely comprehended hereditary factors. The entire "family" in the phyletic sense will have been surveyed, and the variable factors somewhat analyzed.

PROGRAMS FOR RESEARCH

Certain broad programs of research have been systematically planned and magnificent ideals erected for the broad comprehensive study of the human nervous system from all possible avenues of approach. Such may be glimpsed in the work of the Herricks of this country, of the Vogts of Germany, of the Dutch school led by Winkler and ably supported by Kappers, Brouwer [9] and Magnus and others. So far as our particular issue may be outlined, the work of the comparative anatomist, as well as of the embryologist, has indicated phylogenetic and ontogenetic modes of approach which may be followed to advantage.

From the clinical side alone, Brouwer makes interesting suggestions as to the value of these ideas as he has applied them. Speaking of multiple sclerosis, he says, the "comparison between the clinical and the anatomical results in multiple sclerosis" leads us to ask, "How can it be that in this disease, in which the central nervous system is so irregularly strewn with foci, some regularity in the clinical picture can still be obtained?" Then, although multiple sclerosis is rich in forms and variations (paralleled in a number of the hereditary disease forms), one cannot deny that a certain group of symptoms dominates in this disease (picture). These are especially the spastic paresis of the legs with increased reflexes and positive Babinski sign, the disturbances in coor-

9. Brouwer, B.: The Significance of Phylogenetic and Ontogenetic Studies for the Neuropathologist, J. Nerv. & Ment. Dis. 51:113-136, 1920. In Winkler: Festbundel der Psychiatrische en Neurologische Bladen. The implications and applications of this valuable contribution.

dination (ataxia, tremor), the horizontal nystagmus, the dysarthria, the disappearance of the abdominal reflexes. Sensory disturbances, abnormalities in the motility of the eye muscles, bladder and rectal functions remain in the background. They are common, but they are less intensive and more transitory.

Can all these symptoms be considered from one standpoint? He believes they can, and in the manner we have already intimated, for he puts the motif of our own issue somewhat as follows: *"If there is one part of the body of vertebrate animals in which the great line going upward in the direction of man can be seen, it is the nervous system."* The great mass of facts which has been collected of late years by workers in the comparative anatomy of the central nervous system, has gradually convinced neuropathologists that an exact knowledge of the relations in lower animals has also an important value for the insight into the morphology and physiology of the human nervous system. Certain ontogenetic studies are of even greater importance than phylogenetic ones. Our knowledge of ontogenetic problems is, however, far from complete, and phylogenetic studies may elucidate many points which hitherto have remained unexplained.

The advantage of tracing things back to lower forms which are more simple and therefore more clear, exists, not only for the morphologist and the physiologist, but also for the pathologist. This is not sufficiently understood by pathologists. Several facts, found by different workers, show that the more recent acquisitions of the central nervous system are less resistant to noxious agents than older parts. [Not always so, so far as heredity is concerned, as C. and O. Vogt point out.]

BROUWER'S PRINCIPLE IN MULTIPLE SCLEROSIS

This has been shown by a number of workers, and Brouwer would apply this unitary principle to the comprehension of certain clinical facts found in multiple sclerosis (*paralleled in certain heredo-disease pictures*):

Point 1. A woman with multiple sclerosis had never shown other disturbances in the motility of the cranial nerves than a slight paresis of the left facial nerve. Speech, however, was almost totally impossible. In this case I found the brain stem richly involved by sclerotic foci, not only in the nuclei of the cranial nerves, but also in their roots. One may say: It is not astonishing that the cranial nerves have not shown more paresis clinically, because we know that the axis cylinders are saved in the sclerotic area and the stimuli can pass. This is right, but *why then could not the patient speak?*

Point 2. Loss of the abdominal reflexes is a regular and early symptom of disseminated sclerosis. Must it be explained by the fact that this reflex goes partly over the forebrain and that these communicating fibers are interrupted? But the knee reflex also has a communication with the forebrain and *why does*

*this reflex not disappear with the same regularity as the abdominal reflex, when
there are so many sclerotic foci in the thoracic and lumbar part of the spinal cord?*

Point 3. In this case nystagmus was present. Anatomical research teaches
that there are many foci in the medulla oblongata that can explain nystagmus.
But, *why was the nystagmus only horizontal, and why is this horizontal nystag-
mus obtained by moving of the eyes sideways so frequent in multiple sclerosis?*
And why do these sclerotic areas not cause a permanent palsy of the sixth or
seventh nerves, since anatomically there are so many occasions for this?

Point 4. In this case an almost total transverse lesion of the thoracic part
of the spinal cord was present. The palsy of the legs was almost total, but the
patient reacted until death, to pin pricks on the legs, while also tactile stimuli
were perceived. *How shall such a contrast between the motor and sensory dis-
turbance be explained?*

Point 5. In the majority of the cases with atrophy of the optic disk, only the
temporal half is pale. The nasal half remains normal (symptom of Uhthoff).
When one now examines the observations of the anatomic alterations in the optic
nerves, a diffuse lesion is always found. The sclerotic foci are irregularly spread
through the nervi optici and the tractus. Sometimes even the whole transverse
section of the nerve is filled with sclerotic tissue, while clinically only the tem-
poral half of the disk is pale. It does not seem astonishing that the nasal
half is not degenerated, since the axis cylinders are saved in this process and
it is therefore not necessary that a secondary degeneration is caused. This is
correct, *but why then does the temporal half become pale?*

I will now let other difficulties rest, but only add to these examples the inter-
esting fact that the same clinical picture as disseminated sclerosis is also found
in other diseases where the medulla oblongata and the medulla spinalis do not
show sclerotic foci. The most striking is "lobar sclerosis." The pathologic
anatomy of this disease corresponds with that of multiple sclerosis, but the
sclerotic foci are larger and found only in the hemispheres of the forebrain;
it is difficult to explain why the clinical pictures of these diseases show such a
great resemblance, while the regional extension of the process is so different.

It seems possible to reconcile these difficulties by looking at it from the point
of view of evolution. One must admit that the older parts of the central
nervous system, wherein primitive functions are regulated, have greater resist-
ance to noxious agents (through hereditary mnemic usage) than the phylogenetic
and ontogenetic younger parts wherein the higher (later) functions are localized.
If we first look at Point 1, it is clear from this point of view that the tracts
that conduct the function of speech are severely damaged in their function, while
the tracts of the cranial nerves for the more simple movements remain normal.
The first are the so-called phonetic tracts that descend from the forebrain to the
motor nuclei of the oblongata and also the cerebrocerebellar tracts. That these
systems have little resistance is not astonishing, since phonation is a very high
function, which appears only late in phylogeny as well as in ontogeny.

The same idea can be followed for the abdominal reflexes. Generally the
term "reflex" is used for mechanisms of lower order. But there are several
reflex movements which are the expression of a higher organization and appear
very late in phylogeny and ontogeny. Among these is the abdominal reflex,
which only occurs in primates, while the knee reflexes are found in several lower
mammals. The abdominal reflexes appear late in ontogeny (Cattaneo,
Bychowski). The knee reflex is present at birth, but the abdominal reflexes do
not appear until after several months. The latter are phylogenetically and
ontogenetically younger and less resistant.

In this way we can also understand something concerning the frequent occurrence of horizontal nystagmus. The symptom is explicable because of the foci in the medulla and especially because of those in the region of Deiters' nucleus and the fasciculus longitudinalis posterior. But when we wish to understand why the other symptoms, depending on foci in this region, are missing we must admit that not every *part* of the medulla is reacted on in the same intensity by the noxious agents. Horizontal nystagmus, on lateral moving of the eyes, is a very early and regular symptom in this disease. We can understand this fact when we consider that the sideward movement of both eyes at the same time in the horizontal plane is a function which is only present in higher mammals, in which the eyes are placed more in the front of the head and in which the structure of the face makes it possible that such a function has acquired a great significance.

Concerning Point 4, it is an often stated fact that disturbances in motility in multiple sclerosis are much greater and more frequent than those in sensibility. The spastic paresis in almost every case of sclerosis multiplex has some peculiarity that is not found in pure spinal cord diseases. In the majority of the cases it is accompanied by a disturbance of muscle coordination. The paresis is a resultant of damage to the pyramidal tracts. These are phylogenetically and ontogenetically young, especially the part for the legs, and we can understand that this function suffers frequently and early.

The spasm is caused by the circumstance that the phylogenetically older subcortical motor tracts preponderate in the function of motility. With regard to disturbance in coordination, it is not superfluous to remember that this is also an early and very persistent phenomenon. Meijer some years ago published a valuable paper about this fact. He showed after an accurate investigation of a large clinical material that a slight trembling, a slight disturbance of the equilibrium and a slight disorder in the right cooperation of the muscles of the trunk and the extremities are very frequent in early periods of this syndrome. They are often found in cases where the force of the movements is still normal. Several authors are inclined to ascribe these disturbances of coordination to the cerebellum. It seems to me that this is not justified; for, although the researches of late years have taught that the cerebellum is more frequently affected than Charcot believed, this is yet too rare to explain such a frequently appearing symptom as this disturbance of coordination.

For a better insight into this situation, we must study the cerebrocerebellar tracts in the pons Varolii which are important conductors of this function. It is generally known how this part of the central nervous system grows enormously in higher mammals, and these system developments, especially in man, are responses to the erect posture. The same can be stated ontogenetically since the cerebrocerebellar tracts have a late myelinization, much later than the pyramidal tracts. The great number of foci regularly found in these areas makes it comprehensible that an injury to these young systems gives a disturbance in the function depending on these tracts.

As far as sensibility is concerned, I have demonstrated in another paper that there must be a great uniformity in the anatomic organization of the sensibility in the medulla spinalis and the medulla oblongata of vertebrates. The systems for conducting the touch, pain, heat, and cold stimuli must be organized in the lower animals almost in the same manner as in man. They are the phylogenetically older systems of sensibility. The stereognostic and discriminative sensibility on the other hand are recent perfections of the nervous system. It is in the first place the deep afferent system, through which we gain our knowledge concerning

the posture of the limbs and the power of recognizing passive movements, and it is in the second place the system for the function of tactile discrimination of two points, which are more highly developed in the primates. These are the phylogenetically younger systems of sensibility.

We know that disturbances of sensibility in multiple sclerosis are rarely intensive and persistent. In the views defended here we must expect that the sense of deep sensibility and the sense of tactile discrimination are oftener disturbed. In regard to deep sensibility this is true, as Finkelnburg has shown, and it is proved by my own researches in multiple sclerosis. In many cases I found that only the deep sensibility sense was damaged. Tactile discrimination of two points is still insufficiently examined in these cases. Researches in this direction are very difficult, since patients with multiple sclerosis are very often slightly deteriorated, and therefore are insufficient for fine tests.

From the same standpoint it seems also possible to find an explanation of Point 6, the symptom of Uhthoff. We must ask, what is the essential difference between the nasal and the temporal half of the optic disk at the moment that the optic nerve penetrates the eyeball? It is known chiefly through the researches of Henschen that the nasal half of the optic nerve is composed of fibers, all of which cross. In the temporal half we find the fovea bundle and the fibers that run uncrossed. In animals lower than mammals, the optic nerve is totally crossed. This is partly the consequence of their lower organization, chiefly because of the fact that the eyes are standing far sideways in the head.

In lower mammals, where the eyes also stand far sideways in the head, the number of non-crossing fibers is still small. Thus in the guinea-pig the number of non-crossing fibers is insignificant (von Monakow), also in the rabbit (Bach) and in the horse (Dexler). In the carnivora the number of non-crossing fibers is augmenting. The difference between the cat and the dog is striking; in the latter the number of non-crossing fibers is smaller than in the former, which can be understood from the difference in the placing of the eyes in the head (von Gudden). The number of direct fibers is great in primates, especially in man. But in phylogenesis there can be found a higher development not only of the direct fibers, but also of the fovea bundle. It is well known that the macula lutea in man shows a high degree of perfection. The macula has a fovea, and this region is more richly provided with ganglion cells than the remaining part of the retina. In the animals this relation is more simple. [Here a later study of amaurotic family idiocy finds some principles of much importance.]

Among mammals a real fovea is present only in primates. G. Fritsch has shown that the presence of a real fovea is doubtful in prosimiae and is lacking in lower mammals. In the cat this spot is only represented by a little eminence, exhibiting a slight thickening of the layer of ganglion cells (Ganser). In non-mammalia a fovea occurs only in birds. Birds, with their enormous visual faculty, stand apart in the series of vertebrates and in regard to the fovea they also make an exception. Although these relations are not yet sufficiently examined, it is probable that the fovea bundle in the lower mammals—even if this exists—is represented only by very few fibers, and certainly is much smaller than in primates.

Concerning the phylogenetic evolution of the optic disk, it appears that two alterations take place. A large group of direct fibers appear, and the fovea bundle increases. There is no reason to assume that in different animals these fibers have any other course than in man. Thus in the cat the same course of the direct fibers has been demonstrated (Ganser). They take their origin in the temporal half of the retina and remain in the lateral part of the optic nerve.

Consequently the phylogenetic alterations are found in the temporal half of the disk, the nasal half remaining unchanged.

This atrophy of the optic nerve must be considered partly on the same level as the slight atrophy and degeneration of the pyramidal tracts, which is very often found in disseminated sclerosis. It is only in this very young system that we find this alteration, just as in the optic nerve. The atrophy of the disk must be in part a direct result of the degenerative changes of the optic nerves. In regard to the disturbance of vision in multiple sclerosis, it is not possible to give any rule. This can easily be understood when one considers the anatomic relations. The alterations of the optic system by the sclerotic foci not only occur in the nerves, the chiasma and the tract, but also between the corpora geniculata externa and the regio calcarina. One finds regularly severe alterations in the geniculo-optic bundle along the ventricle. We therefore may not expect any regularity in the disturbances of vision, especially not since these groups of alterations are very often present together. It may be remembered in this connection that already in early stages of the disease central scotomas frequently appear, disturbances thus which are caused by a lesion of the fovea bundle, which—as we say—appears very late in the series of mammals.

It appears from this that when we start from the evolution of the nervous system, we have a thread which can help us to find an agreement between the clinical and anatomic phenomena exhibited by this disease. Although the noxious agents in disseminated sclerosis damage of course also older and more primitive functions, such lesions stand in the background of the complex, and they are less intensive and not so persistent as the others.

The fact that by preference the higher, and the latest acquired, functions are disturbed, is also available for the psychical alterations. The researches of late years have shown that the cortex of the forebrain is more gravely affected than was formerly thought. The histologic alterations are not so striking, while the glia fibers in the cortex have not such an inclination to grow excessively as in other parts of the central nervous system. Although these modifications in the cortex of the telencephalon vary greatly in intensity and in localization, the psychic phenomena of these sufferers are rather monotonous. The typical mental image of the patients with disseminated sclerosis is a slight dementia, with a shrinking of their sight sphere and of their interest, a childlike feeling of security, a defective control of their affective life. Although still more severe alterations of the psyche may occur, these remain exceptions. It is very common that the mental condition of these sufferers is reduced to that of the child, to the so-called "puerilisme mentale."

Brouwer applies the same valuable point of view to peripheral nerve function as seen in median nerve neuritis, where the material some day concerning the hereditary muscular atrophies and dystrophies may advantageously be analyzed by some such mode of approach.

His final paragraph says:

It cannot be expected that this rule, i. e., that younger parts of the nervous system are less resistant to noxious agents, is always immediately clear by the findings which one meets in pathology. For there are always several causes acting together in pathologic conditions: viz., heredity, specific affinity of the noxious agents to definite parts of the central nervous system, the difference of the "portes d'entrée," the different intensity of the providing bloodvessels, etc. Thus it is as yet unknown why the noxious agents in poliomyelitis have such a

preference for the gray matter. It seems at first sight that the above-described rule is not followed in this disease, since the larger cells of the gray matter are the most affected and these groups of cells belong partially to the oldest parts of the spinal cord. But Stärcke remarked how the process of poliomyelitis acuta anterior attacks by preference the cervical and lumbar enlargements. Paralysis of the trunk muscles is seldom seen. The pathologic anatomy of this disease has also taught that the alterations in the thoracic part of the spinal cord are far less intensive and less frequent than in the cervical and especially than in the lumbar enlargement. The muscles now of the trunk and their cells in the anterior horns of the spinal cord have undergone in phylogenesis and in ontogenesis almost no differentiation. The muscles of the extremities on the contrary show a great development and variation in their evolution, especially those of the legs, which must be accompanied by great alterations in the cell groups of the spinal cord. Assuming a special preference of the noxious agents of poliomyelitis acuta anterior for the gray matter, it is thus in the light of evolution clear why these parts of the spinal cord are more especially attacked. It is therefore necessary to be very careful in the application of this rule and we must not deduce from the fact that in concrete cases it is not directly clear, that this rule has no further significance.

If from the phyletic point of view it seems a relative fiction, logically valuable, that the latest added structures, either seen as gene mutations (i. e., inexplicable as to where they came from, as new creations) or as gene acquisitions through functional mnemic accumulations (Lamarck, Butler, Semon, Kammerer), are the more susceptible to modification by heredity (apparently provable for the phänotype, less so for the genotype), then it would appear to be logical to attack these neuro-psychiatric variations of apparently simplest form; that is, first make an effort to arrange such in terms in which the fewest genes seem to be involved in the event; then, if possible, apply the apparently most stable of the mendelian fictions. With this last construction perhaps one is still far from the mark; for not many years after dominant and recessive were formulated as mendelian laws, their inadequacy to deal with the behavior of things became evident.

A previous generation of geneticists spoke of direct inheritance in much the same terms as one speaks of simple dominant inheritance. Perhaps one is justified in thinking of it as apparently the simplest.

Ascendant and descendant family trees are to be utilized to the full. Ascendant family trees have the great advantage in showing the possible relationships of the human problems to those of the inbreeding and cross breeding of comparative biologic studies. The family tree, how-ever, becomes an extremely interesting theoretical statement, for by simple arithmetical calculation it may be seen that nine generations back every one of us had 512 ancestors, and in going back to the beginning of the Christian era, each one of us contains the possibilities of inheri-tance of eighteen billion ancestors. Incest then is no problem raised by phantasy, and the Oedipus complex, one of the forms of its expres-

sion, is not a fiction either as portrayed by Sophocles, Shakespeare, Schiller, or Freud. As has been abundantly shown, and will be emphasized here, the incest problem, whether as seen in the heredodegenerations or in the psychologic study of our mnemic patterns as revealed by the investigations of the unconscious, has attained the preeminence, in logical construct, of a theory. It has passed beyond the fictional and hypothetical construct stage (Vaihinger [10]).

A further difficulty of our problem is that of the genes or factors, which it may be necessary to analyze. Plate has emphasized and partly elaborated this theme. If in the sixty generations of historical records we have so many billion ancestors, eliminating the enormous necessary amount of inbreeding (incest), it would look as if the pursuit was a chimera. And so it actually is if we should for the moment *assume a solution,* but if we *know* that we are dealing with fictional thought processes, the undertaking assumes another form, and we may, by the aid of the hereditary discipline, learn some valuable lessons and incorporate them into some generalization which will be of service to ourselves, if not to future generations. Even if expressed in metapsychologic terms, at the worst such delusional constructs—i. e., delusional so far as the mass is concerned—may be of great value to the individual making them.

The peculiarities of human hereditary studies lie in a number of factors. In the first place, the experimental studies of animals and plant crossings which have been widely made are not possible in human heredity. One is compelled to study family trees as far as possible, and such family trees are imperfect and difficult to obtain, especially as one goes back to the second and to the third generation. The desirable or even necessary statistical methods for establishing any hereditary "*laws*" become all the more difficult of application.

Furthermore, as Hoffmann most recently and many others previously have pointed out, the student of heredity in human problems is at the disadvantage of not being able to establish the genotype (Johannsen), structural crossings with which an exact biologic insight is to be obtained. Nevertheless, it is incumbent on us to make use of fiction, hypothesis and theory, representing, in the language of Vaihinger, three advancing stages of mental functioning for the logical solution of the causes of events in order to take advantage of related fictions, hypotheses and theories which have seemed of definite service to the biologist working with infinitely simpler material. As Mendel's observations made older thought functions of little value as generalizations, the past twenty years have shown that the so-called mendelian "laws"

10. Vaihinger, H.: Die Philosophie des Als Ob, Ed. 7 and 8, Leipzig, Meiner, 1922.

are but crude outlines of fictions, hypotheses and theories. It is clearly evident that one must abandon the pathway of absolute idealism and resort to as empirical a realism as the material at hand will permit.

Where shall one begin in such empirical searchings? It is useless to stick to our titular division of "dominant" and "recessive," since we have seen that this generalization has long since been passed. Therefore I shall first discuss those variations of human capacity known as *talent* or *genius*.

HEREDITY IN MUSICAL TALENT

As musical talent may be said phyletically to be of most recent origin, I shall first turn to this field in Haecker and Ziehen's [11] latest contribution, wherein the genealogic method is followed. Here in terms of functional factors are to be distinguished receptor (sensory stimulus) component (optic and auditory), retentive components (mnemic inheritance), synthetic components (ekphoric binding), motor complex (pathway of discharge) and ideative component, each with their as yet incompletely analyzed structural substrate, yet ideally reached for in Vogt's ideal of cytotectonic and myelotectonic brain analysis.

None of the mendelian hereditary types are absolutely followed according to this recognizedly insufficient, yet for the time being most complete, analysis of musical talent inheritance. The modified Pisum inheritance type seems to be most closely followed. Other types, Zea, Avena, Dorset-Suffolk, Abraxas and Drosophila are to be rejected.

So far as negative correlations are concerned for males, musical talent and mathematical talent seem most incompatible, and persons with depressive phases of the manic-depressive types in larger numbers seem to possess musical talent.

INHERITABLE MENTAL DISORDERS

Concerning *inheritable mental disorders,* the situation is not much more promising. With the exception of serious difficulties in clinical conceptions concerning the various psychoses, it must be assumed that the mental functions are correlated with and in some sense dependent on structural substrates in the nervous system as well as any other structural organizations of the body. Such structures undoubtedly behave hereditarily just as other structures behave, even if they do possess the complexity which has been emphasized. As Rüdin [12] has stated, fundamentally there should be no essential differences other than

---

11. Haecker, V., and Ziehen, T.: Zur Vererbung und Entwicklung der musikalischen Begabung, Leipzig, J. A. Barth, 1923.

12. Rüdin, E.: Zur Vererbung und Neuentstehung der Dementia Praecox, Springer, Berlin, 1916; Ueber Vererbung geistiger Störung, Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:459, 1922.

those found in other animals, and the mendelian concepts open up for the psychiatrist new vistas of much importance. The general validity of the principle of segregation is no longer doubted. In human inheritance, however, the difficulties (already outlined) must be borne in mind. The methods of study must be modified; the obstructions are greater, the advance slower. Nature's experiments are quite different from those of the experimental biologist. Johannsen's concept of the phänotype stands out in bold relief in this particular frame.

I believe it here desirable not to go beyond a few general nosologic conceptions, and I shall limit myself to the groups now comprehended as Huntington's chorea, myoclonus epilepsy, manic-depressive psychosis and dementia praecox psychosis.

Huntington's chorea is the most striking type of so-called simple mendelian dominance—which has been analyzed by Davenport [13] and Muncey as a biotype in which the mental factor and the motor factors, falling together, produce the classical syndrome.

In the more carefully analyzed material offered by Entres,[14] no such biotype has been demonstrated. The structural issues involved so far as our pathologic correlations (Lhermitte and Roussy) are possible, support Entres, since it is readily comprehensible that the now fairly well recognized, but still imperfectly analyzed, striatal involvements must produce their frontal lobe system-bound disintegrations (dementia ?), and the biotype conception is unnecessary physiologically. Just what systems (nervous system *parts*) may be isolated as the essential factors (idiotype) in this dominant inheritance, must be left for future analysis when we know more about this most complex aggregate of segmental vegetative tropistic discharge systems.

Attention is next called to myoclonus epilepsy (Lundborg). Here the medelian *simple recessive* typus is almost absolute. Unfortunately, almost nothing is known relative to the structural factors. Incest is the striking sociologic event. Here is a field of great promise concerning possible structural analysis of heredoregenerative factors.

There is a great amount of material on analysis of dementia praecox cases through the mendelian conception. Even though our present nosologic conceptions are admittedly in evolution, there is *some* solid foundation. Heredity studies bring out *some type of recessive mechanism*. Rüdin speaks of it as a dimerous recessive type. Lenz supports a dominant mechanism; Hansen and Hoffman a dimerous recessive-dominant mechanism; Kahn predicates a schizoid anlage which is dominant as a genotype. Kretschmer's athletic and asthenic bodily conforma-

---

13. Davenport, C.: Huntington's Chorea in Relation to Heredity and Eugenics, Bull. 17, Cold Spring Harbor, Eugenics Record Office, 1916.

14. Entres, J.: Klinik u. Vererbung bei der Huntingtonschen Chorea, Berlin, Springer, 1921.

tions are correlations of some structural correlate, which Mott [15] undoubtedly would synthesize with some endocrinovegetative factor primarily related to gonadal activity. As yet the successive reflex arc involving the gonadal-viscerospinal-pituitary-thalamic-rhinencephalic-pallial-striatal-rubrospinal pathway units is only dimly envisaged. Here the "parts" involved, so far as their structural unity is concerned, escape definite predication. All that can be said is that the heredity generalizations help in focusing more clearly the integers of a most complex problem.

Concerning those aspects of the pathology of dementia praecox which may be registered in some form of structural alteration of the nervous system, the most painstaking and fundamental investigation would tend to support the view of the Vogts that here is to be found some type of system disease even though such a concept may be difficult to define (Spielmeyer,[16] 1922). The latest findings of Josephy [17] from this angle permit him to state that the findings indicate that one is on the fringe of a system disease at least. The fatty degenerations and the scleroses show a special predilection for single layers; the breakdown of nerve cells is limited to certain laminae, primarily the third, and to a lesser degree to the fifth and sixth, whereas cell layers two and four remain distinctly uninvolved. If it may not be said that the degenerations are systematized, the cells *that are preserved* show definite layers. I am not unmindful of Bolton's and Lugaro's earlier studies along related lines and the fact that the third layer in the frontal region is the latest to develop and seems the most prone to degeneration processes, as seen in a number of "dementias" of Bolton.

The Vogts have shown that certain topistic units of the cerebrum may show elective disease capacity. This they have termed "pathoclisis." They would refer such pathoclises back to hereditary genes.

No analysis of the dementia praecox situation is availing which leaves out of consideration the so-called motor phenomena (organic, in a narrow sense): the catatonic, cataleptic, motor stiffness, loss of spontaneity, perseveratory activities in automatic (occupational) motions, speech automatisms, etc. Such myostatic disturbances are rightly referred to striatal (pallidal) disturbances, and their so-called psychic correlates are striking (see analysis by Bostroem and others), particularly in the defenses set up against paying attention to incoming stimuli (affective ataxia of Stransky, introversion of Jung) because of ineffi-

15. Carlyle, H. B., and Mott, F. W.: Seven Cases of Tay Sachs Disease, Collected Papers, Rep. Proc. Royal Acad. Med., Arch. Neurol. & Psychiat. 5:1, March, 1911.

16. Spielmeyer, W.: Familiäre amaurotische Idiotie, Zentralbl. f. d. ges. Ophthalin u. Grenzgeb. 10:161, 1923 (full bibliography).

17. Josephy: Beiträge zur Histopathologie der Dementia Praecox, Ztschr. f. d. ges. Neurol. u. Psychiat. 86:391, 1923.

cient symbolic delivery (Reichardt [18]—catatonic stupor and edema of the striatum).

Jelliffe and White [19] have attempted to envisage this situation; above all they would emphasize functional (psychiatric) and structural (neurologic) correlates which would welcome the bringing together of cortical and striatal systems; these are as yet incompletely worked out histologically, but the communications of Josephy, Kleist,[20] of the Vogts and others are offering some foundation for unification of divergent aspects. The fact that Josephy emphasizes that not only cortical changes may be present but also striatum-pallidal modifications are histologically demonstrable, is pertinent evidence of their position that the "organism as a whole" must always be considered in any attempt at a philosophical understanding of a disease process, whether it be, for example, diabetes or dementia praecox. It has seemed to us from the beginning that a highly complex series of neuron disturbances must be present in order to bring about such a widespread biologic anomaly as that seen in dementia praecox. Whereas Josephy accents two components, the organic which lies outside of the cortical alterations and the psychotic which is correlated with the cortical, I am disposed to add a third which I have emphasized in the vegetative, which according to Mott's studies show widespread endocrinopathic alterations and which Lewis [20a] has broadened to include a widespread somatic pathology.

As yet we are not in a position to bring these extremely complex series of factors together. It is futile to say that the histopathologic processes seen are the disease, just as it is foolish to speak of them as resultants—function and structures are one, and it may be that the genetic point of view which looks for a specific pathoclisis in fundamental action patterns will aid us in understanding what we may mean by a disease process.

The better one becomes acquainted with the extremely subtle and kaleidoscopic changes of motility resulting from the encephalitides, seen from the neurologic as well as the psychic side, and having their substrate in the striopallidal and related structures, the more one is hopeful of reaching sound deductions, as well as hopeless by reason of their complexity and multiplicity. The studies of Schilder, Kleist, Hauptmann, Gerstmann and a score of others on this subject are available. Just how this material is to be incorporated into our conceptions of the pathophysiology of the schizophrenic psychoses, cannot

18. Reichardt: Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:561, 1923.

19. Jelliffe and White: Diseases of the Nervous System, Ed. 1, 2, 3 and 4, Philadelphia, Lea & Febiger, 1915, 1917, 1919 and 1923.

20. Kleist: Monatsch. f. Neurol. u. Psychiat. **52**:253, 1922.

20a. Lewis, Nolan: The Constitutional Factors in Dementia Praecox, Nerv. & Ment. Dis., Monog. Series 35, 1923.

be entered into here, but the future is filled with opportunity along these newly opened pathways, first resolutely pushed into by Wilson and the Vogts.

The structural complex will not bear the traffic and breaks down. The deep psychologic discipline concerns itself chiefly with the analysis of the traffic, i. e., the cravings—those that appear highly camouflaged in consciousness and less rationalized at deeper levels. The histopathologist must equally analyze the blue print of the action-pattern routes. The geneticist may aid in showing where a bad contractor put in a piece of defective road bed. Thus far they have not answered fully any of the requirements of this simple metaphor.

The manic-depressive psychoses, mendelianly speaking, show characteristic trends toward *some type of dominant mechanism*. Simple dominance does not seem to be revealed, and Rüdin is inclined to favor a trimerous inheritance mechanism in which two recessive and one dominant factor appear, possibly a ten to twelve times dominant polymery. We know nothing positive as yet of the structural integrations, although there are abundant outlying indications that those pathways are somehow involved in which the thyroid sympathetic units, whatever they may be, are in some way involved. It is not improbable that some typically sex linked factor of predominantly female character is, from the heredity aspect, involved—although what this means from the structural aspect is not yet definable. Whether the new conception of a "cycloid" constitution will help is purely speculative, and Kretschmer's pyknotic habitus remains to be explained.

My conclusion relative to essential epilepsy in its hereditary aspect is that here is as yet an absolutely futile problem. I would reject entirely most of the work heretofore done on this problem. I have been able to find only one type of study which I deem of pertinence to my specific problem. That concerns those observations in which hereditary anosmia shows some correlations with epileptic phenomena. The rhinencephalon as a primary receptor apparatus for nutritive tropisms is of great significance. I would call attention to Elliot Smith's studies in this connection, and I believe it a not too remote possibility to correlate ultimately these conceptions with the studies of Alzheimer and others on the well-known Ammon's horn scleroses shown in certain epileptic brains (Wolfenstein, in 166 cases of genuine epilepsy 42.8 per cent., Aliphan, 50 per cent. Ammon's horn changes). Here widespread system involvements are hypothetically possible.

It is a matter of special interest to try to determine whether in amaurotic idiocy the hereditary factor has its structural representation, i. e., whether one can comprehend histologically the heredodegenerations of amaurotic idiocy. Schaffer has occupied himself quite extensively with this problem. He expresses the opinion that the inferior anlage

of the brain is to be seen in the light of a pithecoid organization and an ontogenetic retardation, but his evidence Spielmeyer considers unconvincing, and his conclusions regarding a disturbed cell architecture —a pathotopic—have been considered unsupportable by C. and O. Vogt and Bielschowsky. Spielmeyer also says that no clear indications of the lack of an anlage have been revealed. It is also incorrect to assume that the ganglion cell changes were due to defective developmental factors, as it is clearly seen, especially in the later forms, that the nerve cells are normally formed and only are destroyed after a progressive process. Naturally, however, it is possible that the most minute structures and elementary constituents of the cell may possess a pathologic anlage which at the present time cannot be made visible. As a matter of fact, there must be hereditary factors which bring about the disease and destruction of all of the nerve cells. Schaffer sees the hyaloplasm as the structural substrate in which the harmful hereditary factors are anchored, and in more recent times Marinesco has raised the hypothesis that the inheritance in amaurotic family idiocy is dependent on the condition of the mitochondria. Spielmeyer says that he cannot convince himself of the value of these hypotheses, and with Bielschowsky he is inclined to maintain that one must stick to what may be seen—the infiltration of the fatty substances and the consequent ballooning out of the cell body and the processes.

Anatomic investigation has not offered definite structural situations which permit the outlining of the *parts* affected by heredodegeneration. Bielschowsky would attribute the ganglion cell disease to some disturbance of the vegetative function of the cell. The mechanism which regulates assimilation and resolution must be primarily damaged; the nervous function is involved only after long continuance of the vegetative disturbance. The cause of the vegetative insufficiency of the ganglion consists in the failure or in the diminished action of ferments necessary for the metabolism of the cells. Tschugunoff raises the possibility that the breakdown of the nerve cell elements is intimately related to the disturbed activity of the endocrins, to which everything may be reduced, but to no advantage.

Speaking in terms of the title of this paper, we are here dealing with definite cutting out of a most important series of receptor structures, i. e., certain optic tracts. In accordance with the statements by Brouwer already quoted, this degeneration of the macular system cuts out of the affected nervous system a great number of incoming energy intake possibilities, with the consequent synaptic failures and a widespread nerve degeneration, the clinical expression of which is a low level of intelligence which may be profitably studied by comparison with lower animal behavioristic methods, particularly the Vogt-Spielmeyer types.

The structural cut-outs have been studied especially by Schaffer in a series of monographs, and his pithecoid micromorphology generalization already referred to as well as to the presence of phylectically older structures in the spinal cord—sulcus cylindricus of His, etc. (Karplus).

It is with a certain amount of temerity that I even raise the question: What may be the functional and structural implications of the human nervous system in its phyletic organization to handle the cosmic forces of gravity and of inertia? Through our knowledge of rotation and the labyrinth in man, the experimental work on the layrinthine mechanisms in lower animals and ontogenetic studies of animal embryos, shall we be able to pull apart some day the highly complex inertia handling mechanisms which we find in certain spino-cerebellar-labyrinthine-thalamic, cortical, associated striatal and pyramidal reflexes and patterns which have slowly accumulated since the days when the tide and rotation movements in water and the forces of gravity and inertia were handled by the fish in a much simpler series of mechanisms than are found in man now?

When with these notes in view we approach the large problem of the heredo-ataxias of the various types, whether we are inclined toward the Landsberger-Oppenheim [21] and related views to regard them as but a series of interrelated gradations or whether we choose to adopt the attitude emphasized by Harnhart [22] which would envisage them, particularly Friedriech's form, more in the aspect of hereditarily analyzable unitary disorders, it may readily be seen that the present day confusion is inevitable since our analyses from all the necessary angles have not as yet reached far enough back into the many evolutionary dichotomies of the functional units fused into the structural compounds known as the posterior columns, the cerebellar tracts, neocerebellum and paleo-cerebellum, frontocerebellar tracts, etc. There is greater hope concerning a philosophical attainment along these lines than ever before, and the heredity method of research may be utilized as a thought function, even if purely in its fictional stage. I would call special attention to Barker's [23] interesting study of Sanger Brown's cases of heredo-ataxia:

The microscopic study in both cases reveals marked degeneration in the gray and white matter of the spinal cord, medulla oblongata, and cerebellum. The degeneration is considerably more advanced in Case XVIII than in Case XX,

21. Landsberger, F.: Die Beteiligung des Grosshirns bei der Heredoataxie cerebelleuse, Ztschr. f. d. ges. Neurol. u. Psychiat. **13**.

22. Harnhart, E.: Weitere Ergebnisse einer Sammelforschung über die Friedreichischen Krankheit (Hereditäre Ataxie) in der Schweiz, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:297, 1923; Beiträge z. hered. Ataxie, Schweiz med. Wchnschr. **53**:139, 1923.

23. Barker, L. F.: Description of Brain and Spinal Cord in Hereditary Ataxia, Sanger Brown, Decennial Publications, Johns Hopkins, No. 53.

though the difference is in degree rather than in kind. The degeneration involves in both cases chiefly nerve-cells and nerve-fibers of centripetal paths, including one system of exogenous fibers of the posterior funiculus (*apparently corresponding to the third fetal system of Trepinski*), the dorsal nucleus of Clarke, the direct cerebellar tract of Flechsig in the lateral funiculus and its continuation in the restiform body. In these structures the degeneration is very extensive; in addition there has been some involvement of the dentate nucleus of the cerebellum and the brachium conjunctivum, and probably also of the inferior olive: in the more advanced case there may be a slight diminution in the number of the anterior horn cells, and of the fibers of the nerve-roots.

Only one case from this family has been previously studied histologically, but that one, fortunately, by Dr. Adolf Meyer, who found no circumscribed cerebellar lesion, but made out a marked diminution in the number of cells in Clarke's nucleus, a degeneration of the direct cerebellar tract, and an elective degeneration of the posterior funiculus, especially marked in the pars cervicalis where it involved the medial part of the fasciculus and the most medial part of the fasciculus cuneatus.

In general, then, the lesions in the three cases thus far studied are nearly identical as regards the neurons involved, although there are differences in the extent of the process quite in accord with the differences in clinical symptoms as the cases were more or less advanced. Dr. Meyer interprets the degeneration in the posterior funiculus as a degeneration of the collaterals which connect the lower levels with the higher (cerebral and cerebellar) apparatus comparable to the degeneration of the lateral pyramidal tracts of the lower segments of the cord where the basilar part of the cerebral peduncle and the pyramids of the medulla show no degeneration. Barker is inclined to look on the degeneration in the posterior funiculus rather as systemic; i. e., as involving only certain of the stem fibers and collaterals of the intramedullary continuation of the posterior roots, namely, those corresponding to one period of medullation in the embryo. Recent studies make it appear probable: (1) that the medullation of the fibers of the posterior roots corresponds more or less closely to the medullation of their intrafunicular continuations, and (2) that the fibers of the posterior funiculi which terminate in the nucleus dorsalis are terminals of the main ascending limb of bifurcation rather than collaterals.

It will be of special interest to contrast the Hand family studied clinically by Klippel and Durante and anatomically by Thomas and Roux with the cases described above. In the French family, the lesion involved chiefly the ventrolateral cerebellar fasciculus of Gowers. Meyer's studies and my own show that in the American family described by Sanger Brown, the dorsolateral cerebellar tract is the one which degenerates. The present report, together with that of Dr. Adolf Meyer, makes it clear that the morbid anatomy of the affected members of the family described by Dr. Sanger Brown presents constant features; we now know with certainty the neuron systems principally involved in the individuals who are affected, though we are as yet entirely ignorant as to why just these neuron systems should be picked out.

It cannot yet be said, even taking Schaffer's studies into consideration, that this position has advanced since Barker's study.

If we follow Harnhart, and here the value of genetic analysis in the large may be demonstrated, the Sanger Brown spinal types and Marie cerebellar types may be considered as belonging to a unitary hereditary complex; but, on genetic grounds, they must be separated

from the Friedreich type, as the latter follow a classical *simple recessive inheritance* modus.   Not one of his family trees, he states, shows the presence of the other types.   Assuming the correctness of this position as a genetic principle, the clinical pathologic correlations may be subjected to a much more strict scrutiny.   Possibly through such closer study the discrepancies of genetic generalizations may be resolved which, according to Siemens' summaries, permits these clinical groupings to be considered as: *simple dominant,* Marie types; *irregular dominant,* Brown types; *irregular recessive,* Friedreich; also biotype with *dominant* and *recessive* factors, and also *recessive sex linked* forms.   With these discordant genetic groupings in view, a possible reassortment of clinico-pathologic material may be possible (especially tracing out speech difficulties, and minute structural variations may be pointed out which the present collection of so-called facts—parts of nervous system involved—do not permit).

We can, however, in view of these genetic studies, lean more heavily on the correlations between histopathologic findings and definite reflex arc systems both isomeric and allomeric.   In the familial Friedreich's forms, the outstanding sensory and motor components—partly dismemberable in histopathologic detail—show the later phyletic discrepancies more strikingly than some of the older systems.   As yet no light has been cast on the cerebellar dismemberments, now made more available through Winkler's recent study of the paleocerebellar and neocerebellar degenerations in atrophy cases.

The photostatic and dynamostatic functions subserved through the dorsal and ventral spinocerebellar complexes, phyletically speaking, are now in a fair way of being analyzable.   The preeminently erect posture of man as a late arriving event has called for new overlays in the spinocerebellar, pontile, rubrospinal allomeric reflex systems.   It is in line with Brouwer's study just quoted that a functional dismemberment with some intimations of its structural substrate may be achieved through the genetic setting apart of definite types.

Concerning the structural components which may be involved in this hereditary spinal paralysis group, Schaffer [8] has contributed a thorough study.   He again emphasizes the point of view here outlined that specific units are involved in the hereditary diseases, and that in spite of the clinical variations, unitary pathologic concepts may be reached when the ontogenetic and phylogenetic principles of nerve function evolution are more definitely kept in mind.   Ontogenetically he insists on the involvement of the ectodermal elements; neurons and glia blood vessels are intact.   The ectodermal structures are involved in accordance with their phyletic age, and thus this principle, already illustrated by Brouwer's study, receives renewed emphasis.   The seg-

mental involvements, according to this principle, may be partly analyzed in the sense in which our problem is envisaged. A detailed summary of this paper is unnecessary for our purpose. Schaffer elects to emphasize (*a*) the involvement of the pyramidal tracts from the upper dorsal region downward, whereas cerebrally the pathologic changes are absent; (*b*) the involvement of the columns of Goll from the upper dorsal segments, bulbar to their nuclei; (*c*) a relatively slight involvement of the greater part of the central convolutions; (*d*) a tectonic choice of the cortical pathology which shows itself in the fibrillary changes (Alzheimer) of the third and sixth layers, with some accentuation in the anterior central convolutions, and also a definite progressive atrophy of the Betz cells and a relative sparing of the fifth layer; (*e*) the spinal ganglion changes offer no support to the changes in Goll's columns. The clinically pure spastic heredodegeneraions show a combined picture: a nucleodistal degeneration of the motor and sensory protoneurons (pyramidal system and Goll's column) which in bilateral symmetrical form arise from an endogenous basis.

I feel, after a careful consideration of these findings, that Schaffer has not made sufficient use of the genetic conceptions, for he leaves us in a quandary as to the significance of the differentiations of the ataxic and spastic forms, which Bremer's [24] studies seem to substantiate as showing fundamental differences. With reference to the *spastic spinal cord* heredodegenerations, the phänotype variations of which are extremely complicating, I would direct attention to Bremer's recent valuable analysis which would attempt to envisage the problem largely from the standpoints here outlined.

Clinically considered, we here deal with the Erb types of spastic spinal disease, the familial diplegic types of Freud, amyotrophic lateral sclerosis and related spastic hemiplegic and diplegic disturbances. Rhein [25] has made an important contribution to this problem. Bremer covers much of the ground that I have here outlined and asks with reference to this medley of spastic spinal disorders: 1. Have we an inherited, i. e., idiopathic, disorder? 2. Does the disorder show a simple mendelian factor, or is it dependent on more inherited units? 3. What type of heredity does it follow? 4. Are there characteristics of any kind in a disease with a characteristic hereditary course which permit any conclusions concerning the fundamental, perhaps cytologic, groupings and relationships of the genes—factor couplings?

24. Bremer, F. W.: Klinischer u. Erbbiologischer Beitrag. z. Lehre v. d. Heredodeg. d. Nervensystems, Arch. f. Psychiat. **66**:477, 1922.

25. Rhein: J. Nerv. & Ment. Dis. **44**:15 (Aug. 16); 224 (Sept. 16) 1916.

Since from the clinical groupings the spastic and ataxic cases seem to mirror different trends, Bremer considers it possibly of value to study their family trees, according to the Weinberg criteria, separately. In the paper here cited, he limits himself more or less to the spastic group even though the material that can be collected is not too well adapted for strict logical conclusions.

Here he believes he finds in Morgan's generalization of "factor coupling" an important conception of value in disentangling the various phänotype manifestations of the heredospastic spinal degeneration in which the true Erb type may possibly be reckoned as an idiotype. These forms seem to show some type of dominant heredity, with or without sex-limited characteristics. In a larger proportion of the various phäno-type groups, recessive heredity seems characteristic. Twenty per cent. of these show close consanguinity. The dominant types belong to the less severe, the recessive, to the more severe forms. Thus the recessive forms show more widespread allomeric involvements, with idiocy, eye muscle palsies, and vision disturbances (Schaffer).

Finally, we pass to the last of Bielschowsky's groups, the hereditary muscular dystrophies and atrophies. It would seem that the least complex of these might be the myotonia congenita group, or Thomsen's [26] disease.

For our port of entry into this problem we purpose using the study of Karl Nissen,[27] whose great uncle Thomsen described an affection from which he himself suffered and which was known to him as occurring in many of his own ascendants and descendants, the earliest indication of the description of which he himself attributed to Bell [28] (1832), although the priority of the question rests with the descriptions of Thomsen and possibly with Leyden's (1866) interesting report in 1874.

The differentiations of abortive cases (Pelz), the paramyotonias—myotonia acquisitiva, myotonia paradoxica and myotonia atrophica—are not our concern at present. Nissen has elaborated these trends, and in his discussion of the symptomatologic and hereditary features has made fundamental contributions to the aspect of this interesting type of heredodegenerations, with a record of seven affected generations.

Nissen himself asks concerning this family tree what conclusions may be drawn concerning its dominant or recessive features, and con-

26. Thomsen, J.: Tonische Krämpfe in willkurlich beweglichen Muskeln infolg ererbter psychischer Disposition (Ataxia muscularis) Arch.f.Psychiat, 6:702, 1876.

27. Nissen, K.: Congenital Myotoma, Ztschr. f. klin. Med. 97:58-93 (May) 1923. Rank: Das Inzest Problem im Dichtung und Sage, Deuticke, Vienna, 1914.

28. Bell: Trans. by Romberg, 1832.

cludes in favor of some *type of dominant inheritance*.[29]   Some form of anomaly passes directly from generation to generation, uninfluenced by healthy admixture with the family stem.   The healthy are to be interpreted through homozygote, the sick as heterozygote, inheritance.   The eugenic aspects do not lie within our province; Nissen has dealt with them thoroughly.

As to the structural situations, Thomsen implicated the "cerebrospinal systems" which for our present day criteria are not sufficient. Engel, Sepilli, Schönborn, Seeligmuller, Gessler and Seifert sought the structural bases in quite divergent parts of the nervous system, and Curschmann (1906) essayed to reconcile these in another direction and with Stocker's studies focused on the central ganglia (lenticular region), thus transferring the physiologic-anatomic considerations to the extrapyramidal systems, toward which Stauffenberg's and Thomalla's studies pointed.   Thus, in Nissen's own statement: Is myotonia congenita to be interpreted in some structural hereditary defect of the striatum complex? Close investigation of the general situation of striatum lesions causes him to raise some objections to this hypothesis, even to deny the validity of this generalization.   He finally concludes that the general picture is that of a primary myopathic disturbance, to which Spiller first called attention, and which in later years Schiefferdecker has most thoroughly set forth as follows: (1) hypertrophy of the muscle, (2) sarcoplasmic changes of a specific nature, (3) enlargement of the muscle nuclei, (4) fibrillary changes, arguing for sarcoplasmic disturbances.   With this general position, the most recent studies seem to agree, and in a most exhaustively studied case by Silberberg [30] the only discoverable lesions were in the muscles themselves and were of vegetative nature. So much for the clinicopathololologic correlations as indicating where one should look for the affected *parts*.   In this connection, Rosett's [31] most interesting study may be of some value.   With reference to his family tree he gathers the following facts: 1. With the exception of myotonia and no myotonia, the recorded characters of the first male, as well as those of the first female, formed an unbroken chain which was transmitted as a whole, constituting the general appearance and the mental state of the particular individual.   2. In some of the offspring the general appearance and the mental state of the first male, in others those of the first female, have proved to be dominant.   3. The offspring who have inherited from the first male his general appearance and his normal mental state, have also inherited from the first female the character of myotonia.   4. The offspring who have inherited the general appearance and the abnormal mental state of the first female, have also inherited

29. Nissen: (Footnote 27) p. 75.

30. Silberberg, M.:   Ueber die pathologische Anatomie der Myatonia congenita, Virchows Arch. f. Path. Anat. **242**:42, 1923.

31. Rosett, J.:   A study of Thomsen's Disease, **45**:1-30, 1922.

from the first male the normal state of his musculature.   5. Myotonia is not sex linked in these cases.

The records on myotonia congenita make no mention of the association of any special physical characteristics with the muscular abnormality, with the exception of a large musculature.   Since no general inference can be drawn from a single instance, we are for the present justified in the conclusion that the particular character which offered a ready linkage to the character of myotonia was that of a large musculature.

In the case of the family in question, then, it would appear that some of its members have a large musculature, not because they are myotonic, but because the hereditary factor of a large musculature proved to be a favorable receptor for the hereditary factor of myotonia. . . .

The suspicion has been forced upon the writer that myotonia congenita is caused primarily not by an inherited abnormality of muscular structure or function, but by an inherited sublethal factor which exerts an injurious action upon the neuro-muscular system.   In seven cases the injurious action of this hypothetical agent was sufficient to determine the balance in favor of death.   In other instances the existence of this agent is manifested in myotonia with atrophy of individual muscle fibrils, or of entire muscles and muscle-groups.   In still others it causes myotonia, with muscular pains and a diminution of the tendon reflexes, with or without muscular atrophy.   In others still, especially in cases where the musculature is not congenitally of large volume, the muscular system is spared, and the action of the inherited injurious agent is upon the highest cerebral structures.

Such a conception of the disease might help to explain not only the singular co-existence of psychoses with the muscular disorder either in different members of the same family or in the same person, but the occurrence of the temporary and acquired forms of the disease as well.   If myotonia congenita is caused by an inherited abnormal content of the body fluids, then a temporary myotonia might be caused by a temporary alteration of the contents of the body fluids of a certain kind.   Such a temporary alteration of the body-fluids must have occurred in the cases described by Talma (40) with an acute febrile onset and severe gastro-enteric disturbances; and by Schultze (36) in connection with gastric dilatation, associated with the symptoms and signs of tetany.   The same principle might be applied to the cases where myotonia has occurred in the course of severe disturbances of the central nervous system, such as the case of brain tumor described by Bremer and Carson (5) ; of myotonia associated with athetosis described by Kaiser (22), and of myotonia occurring in connection with syringomyelia, mentioned by v. Frankl-Hochwart (12) and by Schlesinger.

This conclusion does not help our specific inquiry, but if proved to be more generally valid, it should aid us in focusing attention on the development of the splanchnic component in the muscle fiber, namely, the evolutionary history of the sarcoplasmic element in voluntary muscle. We cannot enter this highly complicated field.

MUSCULAR DYSTROPHIES

So far as the larger problem of the muscular dystrophies is concerned, I shall not attempt any general review but go directly to the

study of Weitz [32] on the heredity situations of this conglomerate, since this investigator has gone most exhaustively into the entire literature and analyzed about 670 cases from at least 346 family trees published in 150 different articles. He has given the most complete study of the situation to date. Utilizing the most recent genetic conceptions, he finds that the modus of the heredity tends to follow at least three specific types: simple dominance, simple recessive and recessive sex linked. Many of the reported family trees are too incomplete to afford the proper number relations to establish the modus. He says he has found no case in which an affected parent has all healthy children, and when such do seem to be present, either the children's ages have been below the homochronic level, or a daughter has carried the factors. He also points out that which has been noted for myotonia congenita, that the simple dominant modus seems to result in the less extensive types of involvement. The objection might be raised that the milder cases with dominant inheritance are milder because they have the affected anlage heterozygote, and that the isolated remaining nonreproductive are sicker because in them the affected anlage is homozygote and naturally must have a different substratum from those showing the dominant modus. Some nonreproducing and severely affected cases show, however, the dominant modus. Of the females in whom the diseased factor arises through mutation, only a portion are affected. He also utilizes Plate's conception of inhibiting factors which show a mendelian modus, which seems to play a predominant rôle in the female, but is absent in the stem fathers or stem mothers in which the dominant modus appears. A type which he calls the Nasse type shows the inhibiting factor markedly. The female seems to be always a carrier. Fuller details are found in Weitz's noteworthy study.

Homology and homochronicity are both present and seem to run parallel, although both fictional "laws" are frequently broken. Anteposition or anticipation seems not to be established firmly, although occurring frequently. The fundamental factors (genotype?) are carried by *either* the father or mother sex cells, the modifying factors are carried by *both* mother and father sex cells. Weitz believes that pure cases are not as rare as has been held, and he is inclined to maintain that the conception of admixture with other endogenous disorders is not sound. He thus comes to the position here already mentioned that genetic study affords definite criteria for establishing specific idiotypes and helps us somewhat out of the morass of a single group of the heredodegenerations as conceived of somewhat by Jendrassik, Bing and others.

---

32. Weitz, W.: Ueber die Vererbund bei der Muskeldystrophie, Deutsch. Ztschr. f. Neurol. **72**:143, 1923.

That biotypy or polymery may exist is undoubted, but this is due to the admixture of other specific factors. Concerning this, see the study in which muscular dystrophy and dementia praecox coupling has taken place.

This group is so large and as yet so incompletely differentiated so far as heredity studies permit, that I can come to no conclusions regarding the structural situations. All that can be said of profit (?) at this time is that there is possibly some transmissible factor or factors involving the visceral nervous system which sets up a pluriglandular imbalance through which the muscle metabolites are selectively disturbed. We have no doubt that the rapidly and enormously widening cultivation of the visceral neurologic field will shortly bring us newer points of view, which with the aid of genetic studies may bring about an essentially more logical grouping and a clearer clinical differentiation of this vast conglomerate.

THE CEREBRAL SUBARACHNOID SYSTEM *

CHARLES EDWARD LOCKE, JR., M.D.

Fellow of the National Research Council, Instructor in Surgery

AND

HOWARD C. NAFFZIGER, M.D.

Assistant Clinical Professor of Surgery

SAN FRANCISCO

In the diagnosis and treatment of intracranial infections and neoplastic diseases, one desires more detailed anatomic knowledge of the cerebrospinal fluid system. Indeed, since the great contribution of Key and Retzius in 1875, little has been added to this knowledge, and the present work has been undertaken in a desire to correct this dearth of detailed information.

HISTORY

Galen,[1] father of anatomy, and Vesalius,[2] in the sixteenth century, described a thin membrane surrounding the brain underneath the dura, but neither of them recognized the arachnoid membrane or the subarachnoid space. In fact, the arachnoid was not established as a membrane separate from the pia mater until the middle of the seventeenth century. It was given its name by Blaes (Blasius),[3] an anatomist of Amsterdam (1626-1682), arachnoid being derived from the Greek word ἀράχνη, meaning a spider's web. Soon afterward, Varioli, an Italian investigator, showed that this membrane existed separately even under the base of the brain. In about 1665, the celebrated Anatomical Society of Amsterdam assured themselves of the existence of a separate arachnoid membrane, and Van Horne definitely demonstrated it to his pupils. In Germany, about twenty-five years later, Vieussens[4] described two separate thin membranes of the spinal cord, and in 1697, an anatomist in Holland, named Ruysch,[5] confirmed the extent of the arachnoid over the cortex and base of the brain.

1. Galen: Opera omnia, Ed., Kuhn **111**:710.

2. Vesalius: Opera omnia anatomica et chirurgica Cura, Boerhave and Albini, De Corporis humani Fabrica, Book 7, p. 537.

3. Blaes (Blasius): "Anatomia medulla spinalis et nervorum inde provenientium," Amstelod, 1666.

4. Vieussens: Neurographia universalis, Francofurti 1690, referred to in Magendies: "Recherches physiologique et cliniques sur le liquide cephalorachidien, Paris, 1842.

5. Ruysch, quoted by Haller: Bibliotheca anatomica **1**:1774.

Concerning the presence of cerebrospinal fluid in the subarachnoid space, as early as 1729, Pacchioni [6] expressed the opinion that lymph was contained between the soft membrane and the convolutions of the brain, but furnished no proof. In 1762, Haller,[7] of Lausanne, propounded the theory that a vapor poured over the surface of the living brain and in the ventricles. He thought that the vapor was made by the arteries and in turn absorbed by the veins. The presence of fluid in these spaces he believed was a pathologic condition of condensation of the vapor due to failure of the veins. Cotugno,[8] of Vienna, however, in 1770, first established the fact that fluid was normally present. He discovered it in the spinal canal and later at the base of the brain, and also saw the fluid continue along certain nerves. By experiment he showed that the cerebrospinal fluid of the brain was in direct communication with that of the spinal cord but thought that the fluid was in the space immediately below the dura.

Bichat,[9] in 1802, advocated the theory that the arachnoid was a double serous membrane, identical with serous membranes elsewhere in the body, forming a closed sac, the "arachnoid cavity," in which the cerebrospinal fluid was formed, and he believed that the arachnoid extended into the ventricles by way of a canal which later bore his name. This canal he believed lay side by side with the vena magna Galeni and extended from the arachnoid in the region between the splenium and the anterior portion of the cerebellum to the third ventricle. He injected dye into the ventricles and recovered it in the large spaces at the base of the brain, and also made an air injection under the arachnoid to show its extent. In 1829, Martin Saint-Ange [10] said he believed that Bichat's foramen was an artefact, but in his work he verified the foramen of Magendie, and by injection of colored fluids showed communication between the subarachnoid and the ventricles. In 1839, Knox [11] pointed out the large arachnoid spaces about the base which we now know as basal cisternae. He injected air, ink and colored water into these basal spaces, and finding that they did not pass into the ventricles, advanced the theory that there was no connection between this space and the ventricles.

6. Pacchioni: Antonio Pacchioni regiensis medici et anatomici romani Opera, Editio quarta Romae, 1741.

7. Haller: Elementa physiologiae corporis humani, Lausannae 4:1762.

8. Cotugno: De ischiade nervosa commentarius Viennae, 1770.

9. Bichat: "Traite des Membranes," Paris, 1802; trans. by Coffin, A Treatise of the Membrane in General and Different Membranes in Particular, Boston, 1813.

10. Saint-Ange, Martin: J. Hebd. de méd., Paris 6:97, 1830; 7:379.

11. Knox: Lancet 1:112, 1839-1840.

Although Bichat and Knox both came very near to the truth, it was left to Magendie [12] in 1842 to prove that the cerebrospinal fluid was contained in the subarachnoid space. Magendie, furthermore, showed that the fluid circulated and had a definite positive pressure. He described the extent of the subarachnoid space over the cortex, and found that it continued around the gasserian ganglion and out along the acoustic nerve and along the optic nerve to the orbit. He called the large subarachnoid spaces at the base of the brain "confluents," and mentioned four of them; the cerebellomedullaris, a second located under the pons and between the cerebral peduncles, a third lying behind, above and on both sides of the pineal gland and the fourth in the region of the optic chiasm. Magendie believed that all these various subarachnoid spaces were in open connection with each other.

Cruveilhier,[13] in 1845, agreed with Magendie as to the location of the fluid, as did the German observers, Ecker [14] in 1843, Virchow [15] in 1854, Luschka [16] in 1855, and Bruns [17] in 1854, and the noted Swiss anatomist Kolliker [18] in 1850. The latter authors believed there was fluid in the subdural space also, although in much smaller quantity. They did not recognize, however, as Magendie did, that the different subarachnoid spaces were in free communication with each other, although Luschka suspected it. Even later authors were confused by the idea of Bichat, and it was not until 1870 that Grey's "Anatomy" gave up the idea of a double arachnoid membrane.

In 1875, Key and Retzius' [19] monumental work appeared, which described graphically and illustrated clearly with beautiful lithographic figures the extent of the subarachnoid space. They also made a careful historical study of the subject. Human specimens were injected with coagulating masses colored with Berlin blue, following which the cranium was either cut away or the whole head was frozen and sectioned. This procedure demonstrated many more or less indefinite points concerning the extent of the subarachnoid spaces. These authors described the subarachnoid cisternae of the brain in more detail than Magendie had done, naming seven cisternae: cisterna magna cerebello-medullaris, cisterna pontis, cisterna intercruralis, cisterna chiasmatis,

12. Magendie: Recherches physiologiques et clinique sur le liquide cephalo-rachidien ou cerebro-spinal, Paris, 1842.

13. Cruveilhier: Traite d'Anatomie descriptive, Ed. 2, Paris 4:1845.

14. Ecker: Physiologische Untersuchungen über die Bewegung des Gehirns und Ruckenmarks, Stuttgart, 1843.

15. Virchow: Handb. d. spec. Pathologie u. Therapie 1, 1854.

16. Luschka: Die Adergeflechte des menschlichen Gehirns, Berlin, 1855.

17. Bruns: Handbuch d. pract. Chirurgie, Tübingen, 1854.

18. Kolliker: Mikroscopische Anatomie 2, 1850.

19. Key and Retzius: Studien in der Anatomie des Nervensystems und des Bindegewebes, Stockholm, 1875, First Half.

cisterna laminae cinereae terminalis, cisterna corporis callosi and cisterna ambiens. Besides this they described the extent of the subarachnoid space over the cerebral and cerebellar hemispheres, the prolongation of the subarachnoid space along certain cranial nerve trunks and the connection between the subarachnoid spaces of the brain and spinal cord. They too expressed an opinion that the subarachnoid space extended into the entire velum triangulare.

From the illustrations of Key and Retzius, it is difficult to construct a clear mental picture of the complicated subarachnoid system, but visualization of these spaces and channels in three dimensions can be obtained by the injection of solidifying masses, from which the brain is removed later. We herewith report the results of such a procedure.

METHOD

The corrosion method of Krassuskaja [20] was employed to make a solid cast of the spaces in question. An injection mass of varying proportions of celloidin and camphor dissolved in acetone was employed under 200 mm. (of mercury) pressure, but because of the great viscosity of the injection mass and the caliber of the cannulas, the pressure when it reached the subarachnoid space was actually very much less. For the sake of clearness and contrast, different dyes were added to the injection mass; i. e., for a red stain, "alkanin" recommended by Huber [21] and for the blue stain, a cresyl violet and brilliant green mixture recommended by Marshall [22] were employed. A dye to be used for this purpose must be soluble in acetone and insoluble in concentrated hydrochloric acid.

Dogs killed by ether and fresh human cadavers were used. The skull was perforated over the posterior horns or over the body of each ventricle and a small nick made through the dura. The next step was an occipito-atlantoid puncture with a 3 mm. trocar, as much cerebrospinal fluid as possible being aspirated. A needle was inserted into each ventricle through the skull openings previously prepared. The correct placement of the needles and trochar was verified when injection of acetone in any one of the three needles quickly escaped from the other two needles. This was continued until all of the cerebrospinal fluid was removed as indicated by the return of a clear acetone and not a milky fluid. The celloidin mass was then injected into one ventricle under low pressure and continued until all free acetone had been expelled and the clear injection mass escaped from the needles in the posterior cistern and opposite ventricle. The latter was then plugged with its stylet.

20. Krassuskaja: Reviewed in Engeb. Anat. u. Entwickl. **13**:521, 1903.

21. Huber: Am. J. Anat. **6**:391, 1806-1907.

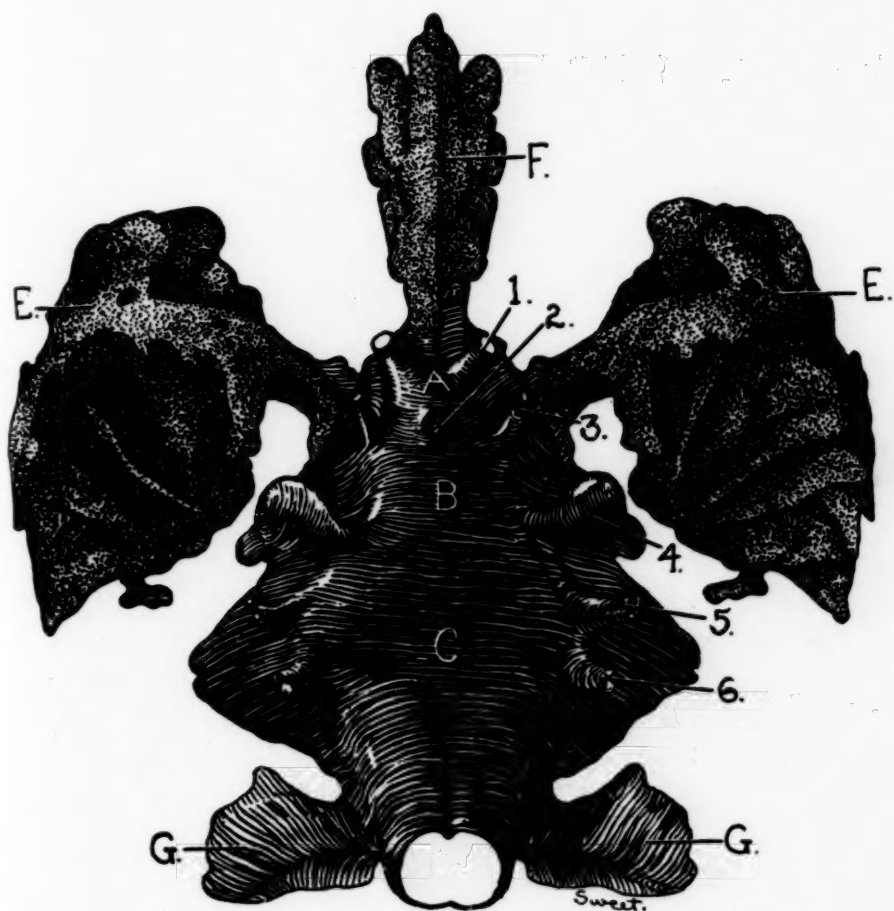22. Marshall, J. A.: Method for Preparing Blue Celluloid Injection Material, J. A. M. A. **80**:181 (Jan. 20) 1923.

Fig. 1.—Basal view of a cast of the subarachnoid space of the human brain. Cisterna basalis, *A, B* and *C*; subarachnoid space about optic chiasm, *1*; about infundibulum, *2*; about oculomotor nerve, *3*; about gasserian ganglion, *4*; about facial and acoustic nerves, *5*; about glossopharyngeal and vagus nerves, *6*; inferior aspect of the lateral cerebrocortical channels, *E;* inferior aspect of the anterior portion of the cerebrosagittal channel, *F*; and the lateral cerebellocortical channels, *G*.
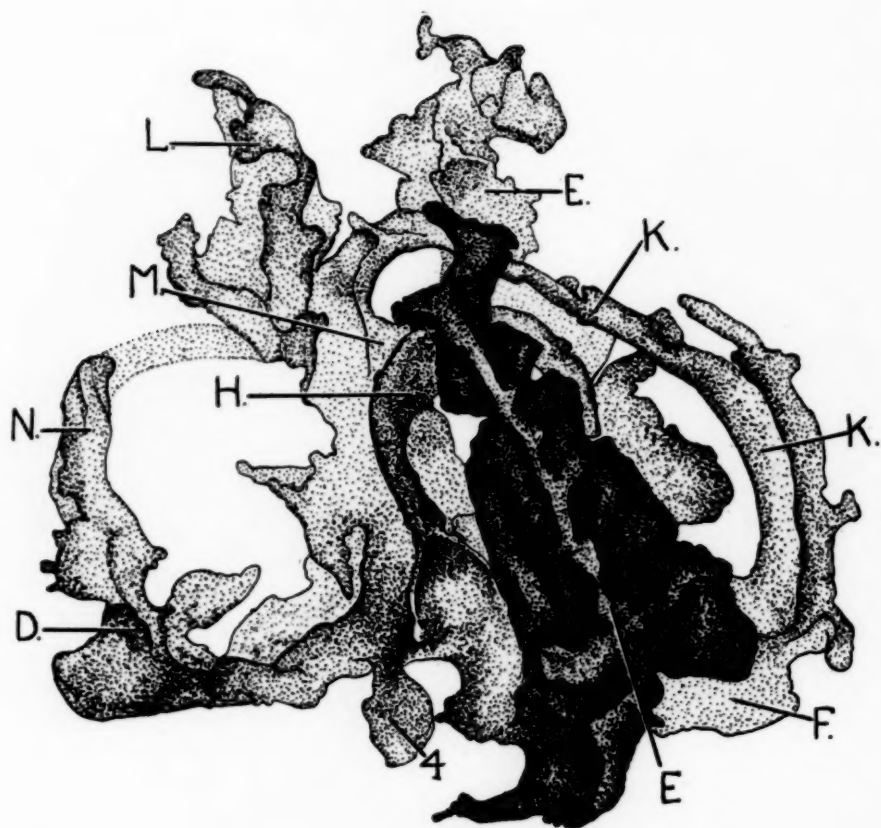
Fig. 2.—Lateral view of a cast of the subarachnoid space of the human brain. The dark colored portion *E* nearest the reader is the right lateral cerebro-cortical channel which partially obscures the cerebrosagittal channel *F*, *K*, *M*, and the internal channel *H*; *D*, cisterna cerebellomedullaris; *L*, occipital sub-channel; *D*, *N*, *M*, cerebellosagittal channel; *4*, gasserian ganglion.
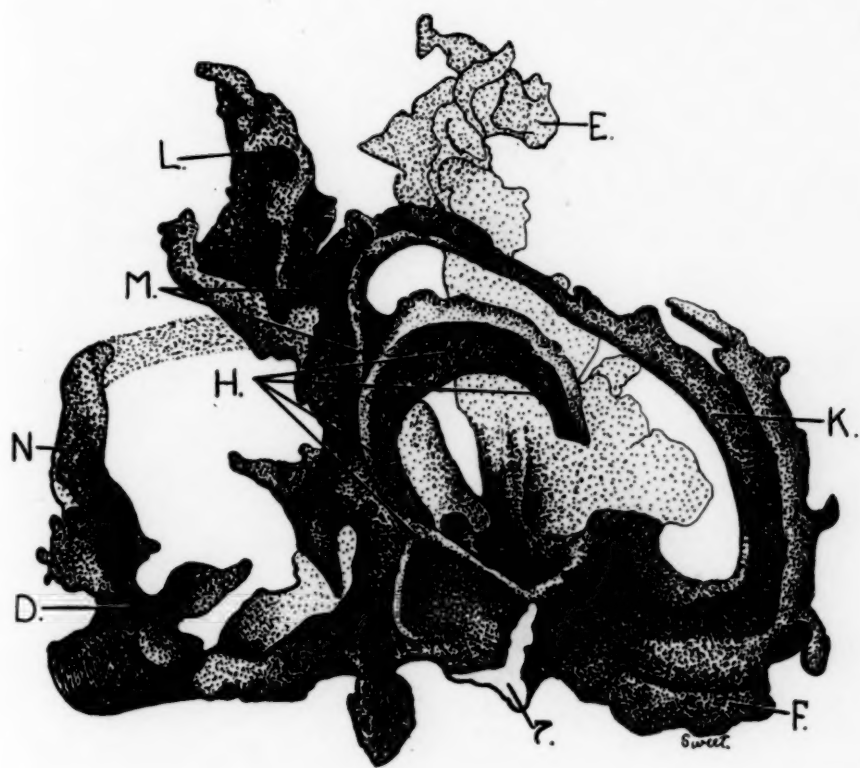
Fig. 3.—Lateral view of cast of the subarachnoid space of the human brain.
The right lateral cerebrocortical channel has been removed at 7, to give a
better view of the cerebrosagittal channels *F, K, M*, and the internal channels, *H*.
D, N, M is the cerebellosagittal channel (in this cast the channel between *N* and
M is imperfect); the subchannel to the right occipital region is seen at *L*; a
lateral view of the cisterna cerebellomedullaris at *D*, and the left cerebrocortical
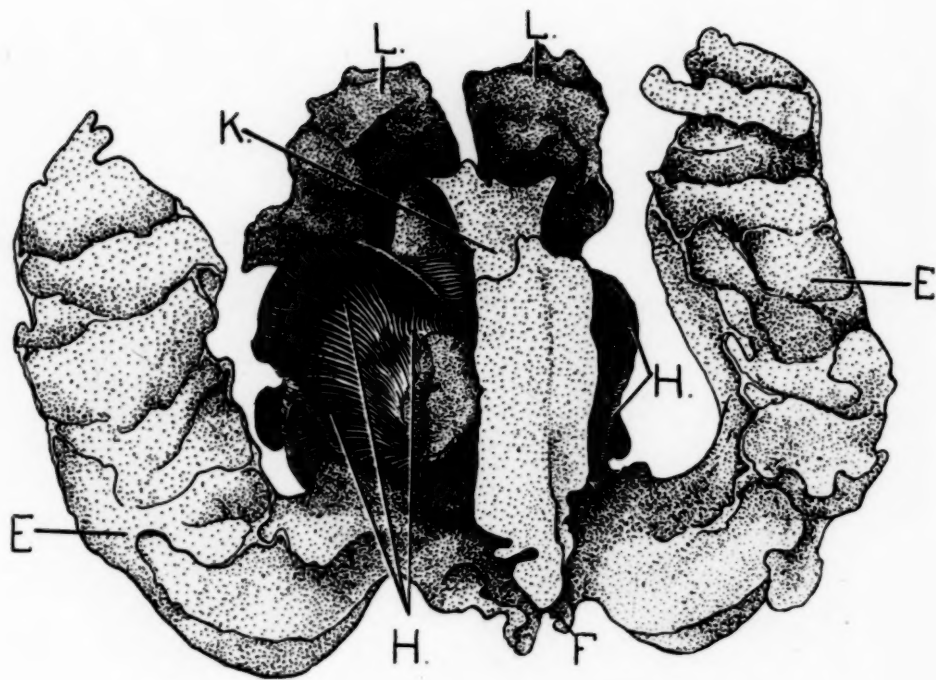channel at *E*.

Fig. 4.—Anterior and slightly lateral view of the subarachnoid space of the human brain. From this angle the right internal channel, *H*, is well seen and also a small portion of the left internal channel; the lateral cerebrocortical channels at *E* and *E*; the cerebrosagittal channel at *K*; and the occipital sub-channels at *L* and *L*. *F* indicates the origin of the cerebrosagittal channel.
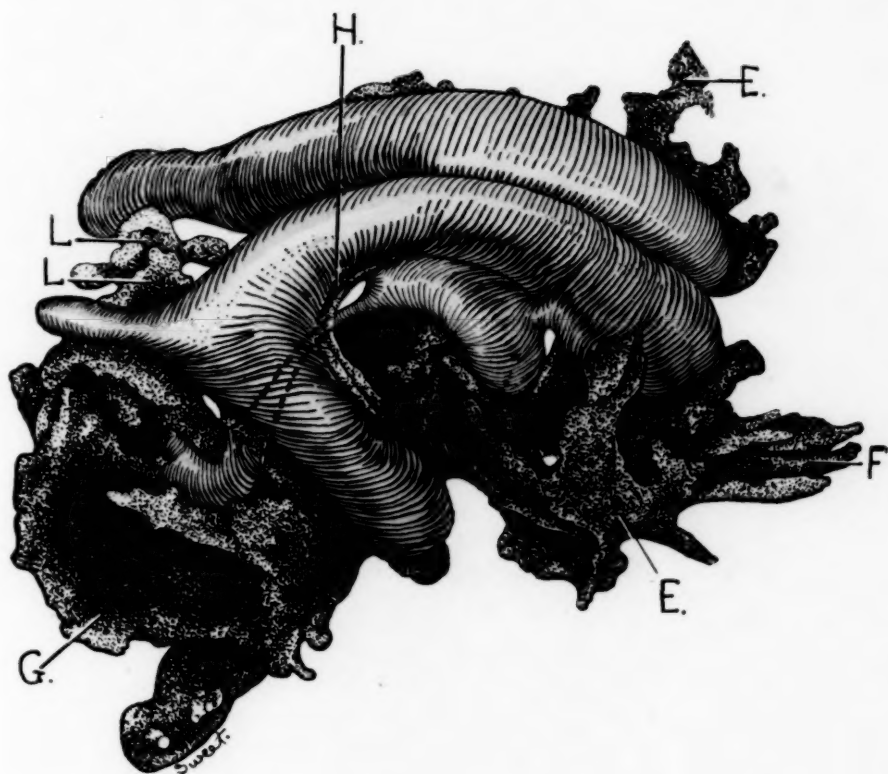
Fig. 5.—Lateral view of cast of the cerebral ventricles and cerebral sub-
arachnoid space. (The cerebrosagittal channel was broken from this cast in
the process of cleaning.) The lateral ventricles, foramen of Monro, third
ventricle, aqueduct and fourth ventricle are in red; the lateral cerebrocortical
channels are seen at E and E; the occipital subchannels at L and L; and a
portion of the right internal channel at H. F, origin of the cerebrosagittal
channel; G, lateral cerebellocortical channel.

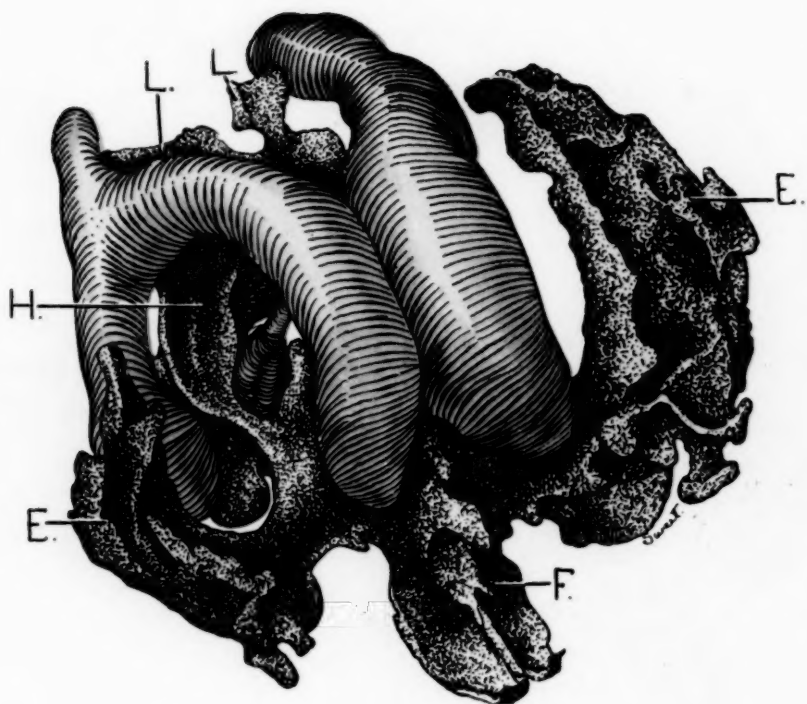Fig. 6.—View of cast of the cerebral ventricles and cerebral subarachnoid space from above and laterally. (The cerebrosagittal channel was broken from this cast in the process of cleaning). The ventricular system is colored red; the lateral cerebrocortical channels are seen at *E* and *E*; the occipital subchannels at *L* and *L*; and a portion of the right internal channel at *H*; *F*, origin of cerebrosagittal channel.

An injection mass of another color was now introduced through the posterior cistern needle and the two injections continued simultaneously. In the ventricle, a 10 per cent. celloidin mixture was first used, and followed by a 20 per cent. mixture, while in the subarachnoid space a 5 per cent. mixture was employed, then 10 per cent. and then 20 per cent. This method of increasing the mass of celloidin was recommended by Morison.[23]

The injections were continued over a period of twenty-four hours, for as the injection mass solidifies by loss of its acetone content, it has a tendency to contract. After the injection was complete, the head of the dog or the cadaver was immersed in cold water for twenty-four hours, and then placed in fresh commercial hydrochloric acid. During the course of from forty-eight to seventy-two hours, the cranium, muscles, dura, and other parts, were removed by the action of the acid, but the brain and stub of spinal cord were left undigested because of their large fat content. The brain tissue itself was removed by repeated washing with a tiny jet of water under pressure, or first softened by the action of pancreatin in 0.5 per cent. alkaline solution kept at 38 C. When the casts had been thoroughly cleaned, they were mounted in distilled water to which a few cubic centimeters of formaldehyd were added to prevent the growth of molds.

RESULTS

By this method, the thinner portions of the subarachnoid spaces are not injected; the principal channels are shown. In the absence of normal circulatory pressure, the brain in the cadaver is more readily compressible, and the main channels as shown in our casts are probably larger than normal. Their general extent and contour, however, are accurate and have given us not only a new conception of the form and extent of the subarachnoid space, but an ability to visualize it not hitherto possible.

The intricate ramifications of the subarachnoid space may best be described by considering the large space at the base and the cisterna magna cerebellomedullaris as the main pathway of the cerebrospinal fluid, and the smaller spaces as channels leading from the main stream. Heretofore, the large subarachnoid space at the base has been described as three cisternae but because these spaces are so completely continuous, we prefer to call the entire space the cisterna basalis (Fig. 1, *A, B, C*) and designate its different portions as pars pontis, pars interpeduncularis, and pars chiasmatis, instead of separating the space into cisterna pontis, cisterna interpeduncularis and cisterna chiasmatis.

---

23. Hinman, Frank; Morison, Duncan M., and Lee-Brown, R. K.: Methods of Demonstrating the Circulation in General as Applied to a Study of the Renal Circulation in Particular, J. A. M. A. **81**:177 (July 21) 1923.

The pars chiasmatis is the portion of the cisterna basalis located farthest forward (Fig. 1). It extends for a short distance anteriorly beneath the frontal lobes in front of the chiasm and posteriorly to fill in the space between the diverging posterior limbs of the chiasm. The pars chiasmatis also extends about the chiasm (Fig. 1, *1*), the infundibulum (Fig. 1, *2*) as well as continuing along the optic nerves toward the globes. In some casts, it partially envelops the pituitary body. The pars chiasmatis, although the narrowest, is the deepest portion of the cisterna basalis.

Posterior to the pars chiasmatis lies the pars interpeduncularis which extends laterally as far as the cerebral peduncles and posteriorly as far as the anterior border of the pons. It is less deep than the pars chiasmatis and from it, collar-like fluid spaces extend out along the oculomotor nerves (Fig. 1, *3*).

The pars pontis lies posterior to the pars interpeduncularis, and here the basal cistern water-bed extends laterally, forming, on each side, a large triangular space with its base at the midline. The apexes of these triangular spaces extend laterally over the anterior aspect of the pons to the cerebellopontile angle (lateral cistern). Here the seventh and eighth pairs, and the ninth and tenth pairs of cranial nerves are surrounded by an extension of the pars pontis (Fig. 1, *5* and *6*), as are the gasserian ganglia (Fig. 1, *4*). Posteriorly, the pars pontis is very much wider than the other portions of the cisterna basalis, yet it is comparatively shallow, being about one-fifth the depth of the pars chiasmatis.

In addition to the cisterna basalis, the cisterna magna cerebellomedullaris (Figs. 2 and 3, *D*) must be included as the second unit of the main cerebrospinal fluid pathway. It lies for the most part in the angle made by the junction of the cerebellum and medulla on the dorsal aspect of the brain stem. Superiorly, it is bounded by the cerebellum and the fourth ventricle, and inferiorly by the medulla. Laterally, it extends out on the inferoposterior surface of the cerebellum and over the lateral borders of the medulla to join the cisterna basalis.

In the posterosuperior direction, the cisterna magna cerebellomedullaris joins the cerebellosagittal subarachnoid channel (Figs. 2 and 3, *D, N, M*), as it extends in the midline over the cerebellum, while inferiorly the cistern continues into the dorsal portion of the tubular subarachnoid space of the spinal cord. The depth of the cisterna cerebellomedullaris is slightly less than that of the cisterna chiasmatis, and about five times deeper than the pars pontis of the cisterna basalis.

From this main cerebrospinal fluid pathway, composed of the midline cisternae, the smaller pathways, which we will call the subarachnoid channels, take their origin. There are seven of these: the paired lateral

cerebrocortical channels, the paired internal channels, the paired lateral
cerebellocortical channels and the single sagittal channel.

The lateral cerebrocortical channels are branches of the pars chias-
matis, appearing in our casts as large moose-horn-like projections
springing from both sides of the pars chiasmatis (Figs. 1, 2, 3, 4, 5
and 6, *E*). They originate just anterior to the inferior surface of the
tip of the temporal lobe, extend anterolaterally along the base for a
short distance and then proceed superiorly and posteriorly following
the contour of the surface of the cerebral hemispheres and lying in the
sylvian fissures. These large channels extend inward between the lobes
and thin out into numerous subdivisions which pass into the subarach-
noid spaces of the neighboring sulci.

The right and left internal subarachnoid channels also spring from
the pars chiasmatis. They are located near the center of the cerebrum
and when injected appear as two leaf-like structures lying in the
interior of our casts (Figs. 2, 3, 4, 5 and 6, *H*.)

From the lateral margin of the pars chiasmatis, on both sides,
these channels extend posteriorly and superiorly, and by joining each
other in the midline, form a well marked subarachnoid arch over the
mid-brain. They then extend anteriorly as a single channel, lying
superior to the third ventricle and partially enveloping the dorsal sur-
faces of the basal ganglia. Superiorly and laterally from them are the
lateral ventricles. Thus these internal channels take their origin
inferior to the basal ganglia, then extend posterior to them, and finally in
the midline are superior to them. As these channels pass inferior to,
then posterior to the basal ganglia, they flare and broaden out laterally,
but as they come to lie superior to the basal ganglia this flare rapidly dis-
appears. At the midline, we find that the internal channel is nothing
else than the velum triangulare, a comparatively large arachnoid tissue
meshwork.

It is important to appreciate that as this space extends laterally from
the velum triangulare, it is separated from the lateral ventricles only
by the fornix and more lateralward only by the thin velum interpositum.
In Figs. 5 and 6-H this close relation is shown. The anteromesial
aspect of the body of the lateral ventricle may be seen to lie in most
intimate contact with this portion of the internal channel.

In the midline just behind the splenium of the corpus callosum the
internal channels join both the cerebrosagittal and the cerebellosagittal
channel which are to be described later. In this region, where the three
channels meet, there is a widening of the subarachnoid space which
corresponds with what previous writers have termed the cisterna
ambiens (Figs. 2 and 3, *M*). From this space fluid pathways proceed
directly toward the occipital region to the subarachnoid spaces over each
occipital cortex (Figs. 2, 3, 4, 5 and 6, *L*).

The sagittal subarachnoid channel (Figs. 2 and 3, *F, K, M, N* and *D*) is not paired, although it consists of an anterior portion, the cerebrosagittal, and a posterior, or the cerebellosagittal channel. The cerebral portion branches off from the pars chiasmatis anterior to and just superior to the chiasma and between the origin of the right and the left lateral cerebrocortical and the right and left internal channels (Fig. 1, *F*). Extending anteriorly and superiorly (Figs. 2 and 3, *F, K* and *M*), it occupies part of the space between the two frontal lobes, then curves around the genu of the corpus callosum, extends posteriorly on its upper surface, and finally curves around the splenium to join the internal subarachnoid channel and there form the cisterna ambiens. The cerebellosagittal channel (Figs. 2 and 3, *D, N* and *M*) [24] proceeds posteriorly from the cisterna ambiens beneath the incisural notch of the tentorium and over the midline of the cerebellum to join the cisterna magna cerebellomedullaris.

The paired lateral cerebellocortical channels (Figs. 1 and 6, *G*) are branches of the cisterna magna cerebellomedullaris and cisterna basalis, and extend out over the surface of the cerebellar hemispheres as do the lateral cerebrocortical channels over the cerebral cortex, although in a diminutive form.

These are the principal channels of the cerebral subarachnoid system. Studies of the finer ramifications of the spaces which extend to the points of fluid absorption are being continued. Proper illustration of such complex casts has been most difficult. Drawings or photographs convey only in part the mental picture to be gained by a direct study of them.

The possible routes for fluid distribution have become evident, and collateral circulation in cases of blocked spaces can be seen. The spread of meningeal infections along the principal channels has certain obvious possibilities. In instances of fluid block, as in hydrocephalus, certain methods of short circuiting of the circulation have appeared, and their practicability is now being investigated clinically. It is hoped that they may prove of practical value.

---

24. Part of the cerebellosagittal channel was broken (between *M* and *N*) in the cast from which this figure was made.

REMISSIONS IN GENERAL PARALYSIS *

A STUDY OF CONSECUTIVE ADMISSIONS OF MEN TO THE MAN-HATTAN STATE HOSPITAL FROM JULY 1, 1911, TO JUNE 30, 1918

MORTIMER WILLIAMS RAYNOR, M.D.

First Assistant Physician, Manhattan State Hospital; Assistant Professor of
Psychiatry, College of Physicians and Surgeons, Columbia University

NEW YORK

This study was made for the purpose of gathering data concerning cases of general paralysis in which treatment had not been given, which could be used as control material for the cases of paralysis in which the patients were being treated in this hospital. The results in the treated series have been presented by Dr. Isaac J. Furman.[1]

The material consists of consecutive admissions of men with cases diagnosed as general paralysis. The period covered is from July 1, 1911, to June 30, 1918, or seven years, during which 1,004 patients, with 1,043 admissions, entered the hospital. Cases in men only have been studied, as the systematic treatment of general paralysis has been limited practically to them. This period was selected because serologic examinations were frequently made, because it antedated the period of the cases in which treatment was given, and because this interval to the present time has been sufficiently long to estimate the course of any improvement that may have taken place.

The diagnoses as they occurred in the case records have been accepted, except as stated below. Each case had twice been considered in staff conference, the initial and the summary presentations. The cases of all patients paroled and of nearly all discharged without parole were considered a third time in staff conference. Patients readmitted were again considered at the conferences. Many had had lumbar punctures and serologic examination, generally for globulin, cells and a fluid Wassermann test. A few had had no Wassermann test made, and a few others had a Wassermann test made on both the spinal fluid and serum. Of the patients who died, 160 were examined postmortem.

No case was accepted in which the serologic examination was negative; in which the serum Wassermann test was positive but the cerebro-

---

1. Furman, I. J.: The Treatment of Neurosyphilis at the Manhattan State Hospital, State Hospital Quart. 8:84, 1922. Treatment of General Paralysis; Results Obtained in a Series of Five Hundred Cases, this issue, p. 359.

spinal fluid negative; in which at necropsy there were not the characteristic findings of general paralysis; and no case was accepted in which the clinical diagnosis had been changed.

Of those patients who left the hospital, the statement in the record as to the condition on parole and on discharge has been accepted. Of those patients who had more than one admission, improvement during any admission has been accepted as such. A few exceptions to this rule have been made. One patient noted as discharged at the expiration of parole as "much improved" was found on inquiry to have been readmitted to another hospital with an exacerbation of symptoms. Most of the records showed that the patients had reported during the parole period, and they contained statements of the patient's progress during that period.

Many of the patients had been transferred to other institutions. A record of the dispositions of these cases has been obtained. The same may be said of patients who had been discharged and readmitted to other institutions.

With the exception of sixty-two patients who were deported, all patients discharged from this or other institutions have been investigated by the social service department as to the progress and outcome of the case and of the present condition of these patients. Letters have been addressed to all correspondents in those cases not located by the social service and through the cooperation of the postal authorities, several patients have been located and their condition ascertained. The friends of patients who had left town were written to, and the patient's condition ascertained by means of a questionnaire. We have failed to find twenty patients. A few patients had had some treatment, but none had had systematic courses.

As pointed out, the condition of the patients on parole or discharge has been accepted as "unimproved," "improved," "much improved" and "in remission," as it is obvious that the opinions of the physicians who had these patients under observation and of the staff conferences were much more accurate than any current opinion could be, viewing the cases retrospectively after from twelve to five years. My judgment does not, therefore, enter into these conclusions. It does, however, enter into the conclusions as to whether a remission had taken place in patients paroled or discharged as improved.

Before undertaking this study, certain criteria were formulated as a guide in estimating whether a patient would be classified as improved only or as having a remission. I shall not raise the question of recovery. The term "improvement" is to be understood as an improvement in the clinical picture during hospital residence which was noted at the time of leaving the hospital, but with a continuation of some of the symptoms to such an extent that the patient was considered abnormal by his

family and friends and was unable to engage steadily in a remunerative
occupation and maintain himself and his family; in other words, he
could not resume his former position in society. The term "remission"
is used to denote what some may call a "social recovery"—a return of
general physical vigor; an absence of ataxia in ordinary movements;

TABLE 1.—*General Paralysis, Untreated Male Cases, Admitted from 1911 to 1918*

	Number	Percentage
Patients	1,004	
Admissions	1,043	
Dead	882	87.8
No recent data	20	
Living	40	
	942	
No data	62	
Total	1,004	
Died in hospital first admission	784	78.0
Paroled, returned, died	20	
Discharged and died	59	
Remission and died	19	
Total	882	87.8
Discharged	117	12.4
Paroled and returned	20	
Total left hospital	137	14.5
Discharged, unimproved	36	
Paroled, unimproved, returned	16	
Total	52	5.5
Discharged improved	48	
Paroled, improved, returned	4	
Total	52	5.5
Remissions	33	3.5
Total improved	85	9.0
Improved, dead	31	
Improved, paroled, returned	4	
Improved, living	5	
Improved, no recent data	12	
Total	52	5.5
Remissions, dead	19	
Remissions, living	14	
Total	33	3.5

coarse tremors and speech and writing defects, although there may be
a persistence of disturbances of deep reflexes and pupillary signs; a
disappearance of all trends and abnormal conduct; subsidence of emo-
tional variations; good memory for ordinary events and happenings; a
certain degree of insight and ability to care for themselves and to engage
in a remunerative occupation and maintain themselves and family in

the community; ability to resume the former status in society. No account is taken of the duration of the remissions of the symptoms, except to note that they are continuous.

Tables 1 and 2 show that of 1,004 patients comprising this study, 882, or 87.8 per cent., are known to be dead; twenty were discharged, concerning which we have no recent data; forty are known to be living; this makes a total of 942 patients who are accounted for or concerning whom we have some data; sixty-two were deported, and concerning them we have no information.

Of the 882 who died, 784, or 78 per cent., of the total number of patients (1,004) were first admissions and died during their continuous hospital residence. Twenty were paroled and returned to the hospital and died; fifty were discharged as unimproved or improved and died; and nineteen who had remissions died.

The 137 patients who left the hospital represent 14.5 per cent. of the total (942). Of these, 117 were discharged—of twenty of these, twelve as improved and eight as unimproved, concerning whom we have no recent data; thirty-one as improved, who died; twenty-eight as unimproved, who died; and five as improved, who are still living; nineteen having remissions died and fourteen are living; and twenty (four as improved and sixteen as unimproved) were paroled from the hospital and returned and died.

The fifty-two patients who left the hospital as unimproved—thirty-six discharged and sixteen paroled—represent 5.5 per cent. of the total. The eighty-five patients (9 per cent. of the total) who left the hospital as improved were distributed as follows: discharged forty-eight, paroled four, totaling fifty-two, or 5.5 per cent. of the total; remissions occurred in thirty-three cases, or 3.5 per cent. The restricted improved group of fifty-two represent 5.5 per cent. and were distributed as follows: dead, thirty-one; paroled and returned and died, four; living, five; and twelve concerning whom we have no recent data. Patients having remissions are represented by thirty-three cases, or 3.5 per cent. of the total 942 cases. Nineteen of these died, and fourteen are living.

Of the total number (eighty-five) of patients who were improved, fifty-two, or 61.1 per cent., were improved only, and thirty-three, or 38.9 per cent., had remissions. Of those with remissions now living, the longest period of hospital residence was three years and ten months, and the shortest period three days, while ten years and one month is the longest period out of the hospital, and three years and two months the shortest. The serologic examinations were found to be positive in all patients paroled improved, in forty-three of the forty-eight discharged improved, in seventeen of the nineteen with remissions who died, and in twelve of the fourteen with remissions living. Two of the four patients paroled and one of the patients discharged improved were

examined postmortem. Unfortunately, no necropsies were performed on the patients who had remissions and died. This is a rather regrettable defect in our material.

The clinical pictures as presented by the mental symptoms in the thirty-three cases reported have been divided into five groups: Group 1, gradual onset, with changes in disposition, dulness, emotional instability, orientation and memory defects; in some cases expansive trends; Cases 1 to 17, inclusive. Group 2, onset of short duration, euphoria with restlessness and expansive trends; Cases 18 to 22, inclusive. Group 3, depression with or without retardation or agitation, associated with worry, feelings of insufficiency or somatic complaints; Cases 23 to 28, inclusive. Group 4, delusions of persecution, poisoning and jealousy; auditory and visual hallucinations; Cases 29 to 32, inclusive. Group 5, gradual onset with change in disposition, emotional instability

TABLE 2.—*General Paralysis—Summary of Outcome*

	Patients	Number of Admissions
Total number of patients...	1,004	.....
Total number of admissions..	.....	1,043
Died in hospital, first admission (78 per cent.)...........................	784	785
Deported	62	63
Discharged, improved, no recent data......................................	12	12
Discharged, unimproved, no recent data....................................	8	8
Living, Manhattan State Hospital..	10	12
Living in other hospitals...	11	12
Paroled, improved, returned and died......................................	4	4
Paroled, unimproved, returned and died....................................	16	16
Discharged, improved, died..	31	41
Discharged, unimproved, died..	28	39
Discharged, improved, living..	5	5
Remissions, died	19	30
Remissions, living	14	16
Total..	1,004	1,043

and delusions that people were against him, with a remission; later developed a depressive reaction followed by a second period of remission; Case 33.

REPORT OF CASES

CASE 1.—A liquor dealer, aged 38, was admitted to the hospital, July 3, 1912, as a voluntary patient. He denied syphilis. He had lost his hair at the age of 19. His wife had had four miscarriages, then one healthy child. He had stopped drinking at the age of 29. There was a gradual onset of symptoms at the age of 36, with drowsiness, irritability, memory and retention defects. He omitted letters in writing. He had unequal Argyll Robertson pupils, overactive knee reflexes, tremors of the tongue and fingers; slurring speech, a positive globulin reaction of the cerebrospinal fluid and seventy-three cells. Wassermann tests were not made. In the hospital, he was assaultive and destructive. After one month in the hospital, he was discharged, Aug. 6, 1912, on request. For nine months he did not work. He helped about the house, then he returned to his liquor business. In 1918, he went to work in his brother's store. At present, at the age of 49, he is working regularly, is self-sustaining and considered well by his family and appeared so to the social worker.

CASE 2.—A theatrical electrician, aged 53, was admitted to the hospital, Nov. 7, 1913. He had had syphilis at the age of 38. At the age of 52, he gradually became nervous. He had an expansive trend as to wealth and his abilities, with failure of memory. Finally he became excited and was admitted to the hospital in an elated, overactive excitement with an expansive trend; he had good orientation and memory, discrepancies in correlation of time relation of past events and poor retention. His pupils were unequal, irregular and sluggish to light. His knee reflexes were exaggerated. He had tremors of the tongue and hands and a speech defect. The cerebrospinal fluid showed an increase of globulin and contained fifty-seven cells. The Wassermann reactions were not reported. Improvement began three months later. He was discharged as improved, May 10, 1914, six months after admission. Orientation and memory were good, retention poor. He was composed, but perhaps too optimistic. After one month at home he returned to work for six months, although his memory was bad. Suddenly his "mind went blank," and three weeks later, Jan. 7, 1915, he was admitted to the Chicago State Hospital, where he remained a bed patient until he died, March 23, 1915, at the age of 54. Serologic examination was reported as "Wassermann positive, spinal fluid 4 plus, sixty-five cells."

CASE 3.—A man, aged 43, was admitted to the hospital, June 25, 1913. He had been an employee of the Edison Company for twenty years. He had had syphilis at the age of 19. Since the age of 35, there had been gradually increasing lessening of his efficiency, with forgetfulness. At the age of 42, he received three doses of arsphenamin. Since then he had been unsteady and excitable, with marked failure of memory; he had to give up work, was ataxic in gait and speech for three months; childish, tearful, afraid to be alone, and thought every one was against him. On admission, at the age of 43, he was dull, childish, indifferent and rambling in speech. He had marked defects in orientation, memory and retention. He omitted letters in writing. His pupils were irregular, equal and reacted promptly to light and accommodation; his knee reflexes were exaggerated; he had tremors of the tongue and hands; his speech was slurring and his gait unsteady. No serologic examination was made. After one and one-half months of hospital residence, he was paroled, Aug. 2, 1913, as unimproved. After three months in the country, he returned to work for his former employer as purchasing agent for stationery, at which position he has been working since. Two years ago, a blood examination by the department of health was negative. At the age of 53, he is well and self-sustaining.

CASE 4.—An actor, aged 52, was admitted to the hospital, Aug. 28, 1913. He had syphilis at the age of 32. He was intemperate. At the age of 42, he was dull and drowsy and did not work. After four years, he was well and was employed for six years. Then he again became dull and idle and gradually lost his memory, became restless and incontinent; after four months, he was admitted to the hospital. He was dull, confused and inactive, with marked defects in orientation, memory and retention; he had no delusional trends. He had Argyll Robertson pupils, exaggerated knee jerks, ataxia, weakness and tremors of the tongue and fingers. His speech was slurring; his blood pressure was 140. The globulin test of the cerebrospinal fluid was positive and there were twenty-four cells. The Wassermann reactions were positive in the serum and the fluid, the latter showing a paretic gold curve. After ten months, he began to show improvement, and after twenty-five and one-half months, at the age of 54, Oct. 8, 1915, he was paroled as improved. He was able to keep track of events and his

general physical condition was improved. He returned to his work as an actor and has since been well, employed and self-sustaining. He was last seen in January, 1923.

CASE 5.—A laborer, aged 44, was admitted to the hospital, Aug. 3, 1914. He had had a chancre at the age of 36 or 37. At the age of 43, he had had headaches for days at a time, when he refused to talk or eat and remained in bed. He became more dull, and after four months he began falling to the floor. This continued up to the time of his admission to the hospital eight months later. He appeared dull and apathetic and smiled in a silly manner. At times he was excited and gesticulated. Orientation and recent memory were good, remote memory and retention were poor; he spoke of being depressed. He had Argyll Robertson pupils, the knee jerks were exaggerated. He had tremors of the tongue and facial muscles. The cerebrospinal fluid showed a negative test for globulin, but it contained 42 cells. The Wassermann test was positive. A serum Wassermann test was not made. After seven and one-half months in the hospital, he was paroled, March 28, 1915, as improved. He went to work directly as a laborer and was employed steadily until a year ago, when he was playfully jostled by a fellow worker, fell and injured his shoulder, which he has since been unable to use. On May 10, 1923, at the age of 53, to the social worker he appeared well physically and intellectually.

CASE 6.—A piano polisher, aged 41 years and 6 months, was admitted to the hospital, May 5, 1914. He had had syphilis, the date of which was unascertained. For three months, he had been absent-minded and had omitted letters from written words. There had been a change in his disposition; for ten days he had been restless and irritable and had had ideas of great wealth. There was no change in his condition following his admission to the hospital; orientation and memory were good; retention and calculation were poor. He had unequal Argyll Robertson pupils; his knee jerks were exaggerated; he had tremors of the facial muscles; the left side of the face was flattened; his gait was unsteady and his speech defective. No serologic examination was made. After three months in the hospital, he showed physical and mental improvement, with insight, although slightly euphoric. He was paroled, June 15, 1914, at the age of 42 as improved. After three months at home, he returned to his former work as a piano polisher; four months later he had a weak spell; after two weeks he returned to work for five months, when on account of weakness and dizziness he remained at home. One month later, July 21, 1915, he was readmitted in an inactive, emotional, unstable state. His talk was rambling, orientation and memory were good, but retention and calculation were defective. Neurologic signs were unchanged. Failure was progressive, and after one and one-half months he died, Sept. 5, 1915, at the age of 43.

CASE 7.—A music teacher and singer, aged 37, was admitted to the hospital, March 7, 1917. He had acquired syphilis at an unknown date. At the age of 33, there was a gradual change in disposition with speech defect. One month after an injection of arsphenamin he became euphoric and expansive about his great wealth, and was admitted to the hospital at the age of 37. Orientation was intact; memory for recent events was good; remote memory was poor, and he was unable to correlate dates. His pupils were irregular and sluggish in accommodation to light. His knee jerks were exaggerated. He had tremors of the tongue, facial muscles and fingers. His speech was slurring. After approximately one month, he was paroled as improved. He had some insight but expressed a feeling of well-being. He resumed his vocation of music teaching satisfactorily for about one and one-half years. Then he suddenly developed a

euphoric state with a markedly expansive trend of great wealth and was readmitted, Nov. 17, 1918, at the age of 38 years and 6 months. He presented well marked defects in orientation, memory and retention. He finally became quiet, and at the end of ten months he was again paroled, Sept. 13, 1919, at the age of 39 years and 6 months, as much improved. At home he helped with the house work and did errands, but his memory was poor. After three years and three months, at the age of 43, he developed confused, irritable and disturbed periods and was again admitted to the hospital on Feb. 8, 1923. At present, he is in the hospital in a restless, poorly accessible state, with marked orientation and memory defects. He has Argyll Robertson pupils, exaggerated knee reflexes, tremors of the tongue and facial muscles and slurring speech. The cerebrospinal fluid showed a 3 plus globulin test, and it contained twenty-seven cells. Both fluid and serum Wassermann tests are 4 plus.

CASE 8.—A longshoreman, aged 38 years and 6 months, was admitted to the hospital, April 19, 1917. He had had syphilis at the age of 20. At the age of 35 years and 6 months, he had numbness and pain in the feet and legs; at 36 years and 6 months, he had weak spells. The spinal fluid and serum Wassermann tests were positive. He received arsphenamin treatment; at the age of 37 years and 6 months, he was irritable; at 38 forgetful, gradually becoming boastful, and had many schemes for making money. Before admission to the hospital, at the age of 38 years and 6 months, he was overactive and overtalkative. Orientation was good, memory for events was good, but there were discrepancies in correlating dates; retention was poor, he also had acutely disturbed periods of short duration. The pupils were irregular and fixed; the knee reflexes were exaggerated. He had tremors of the tongue, facial muscles and fingers; incontinence of urine and speech defects. The cerebrospinal fluid gave a positive globulin test, and it contained 16 cells; the Wassermann test was 4 plus; a serum Wassermann test was not performed. Five months later, Sept. 8, 1917, he was paroled as improved. He worked steadily. Finally he became depressed and was readmitted, April 10, 1918, at the age of 39 years and 6 months, in marked depression, with apprehension and a hypochrondriacal trend, "blood all dried up." He died, June 30, 1918, at the age of 39 years and 6 months, two and one-half months after readmission.

CASE 9.—An iron worker, aged 45, was admitted to the hospital, Oct. 17, 1916. He had had syphilis at the age of 25 or 30. At 41 years of age, he expressed ideas of poisoning; at 42, he began to have convulsions; since the age of 43, he had had periods of forgetfulness and emotional instability. On admission, at the age of 45, he was dull, depressed and had a hypochrondriacal trend, orientation and memory defects, some insight. He had a penile scar; his pupils were irregular and fixed to light; he had exaggerated knee reflexes; tremors of tongue, facial muscles and fingers and a speech defect. The cerebrospinal fluid gave a positive globulin reaction and contained fifty-three cells. He had a 4 plus Wassermann reaction; a serum Wassermann test was not made. Ten months later, at the age of 46, he was discharged as improved. He appeared well and returned to the former employment as an iron worker for approximately six months, when he became confused and assaultive. He was cared for in a neurological hospital for two months, when he was readmitted, May 7, 1918, at the age of 46 years and 6 months, in a dull state, with marked impairment of orientation and memory. The neurologic signs were unchanged, except that ataxia was marked. The cerebrospinal fluid gave a positive globulin test,

and it contained twenty-eight cells; the Wassermann test was 4 plus; a serum Wassermann test was not reported. There was a physical and mental decline until death, Nov. 18, 1922, at the age of 51.

CASE 10.—A painter, aged 34, was admitted to the hospital, April 29, 1915. He had had syphilis at the age of 24. He had drunk to excess at times. For two years he had had emotional upsets. For one week he had been acutely excited, had expressed ideas of wealth and had shown hostility against his wife. On admission to the hospital he was in an elated, overactive, voluble, uncooperative and unclean state and showed the same delusional trend. He was apparently disoriented. His pupils were irregular and sluggish in reaction to light. His knee reflexes were exaggerated. He had tremors of the tongue and fingers. The cerebrospinal fluid gave a positive globulin reaction and contained seventy-four cells. Three months after admission, he began to show improvement. Five months later he was quiet, worked on the ward, and orientation and remote memory were good. He had a poor grasp on events of the period of his excitement. He presented emotional instability and had no insight. Dec. 7, 1915, eight months after admission, he was discharged to go to a private sanitarium. Two weeks later he left and went to work as a painter, earning $5.00 a day. He did well for more than two years. Then his wife left him, and four months later, June 22, 1918, he was readmitted to the hospital at the age of 37. He was in an acutely disturbed condition, had an expansive trend and defects of orientation, memory and retention. His pupils were unequal. There were no other changes in the neurologic signs. The cerebrospinal fluid gave a positive globulin reaction and contained forty cells. He had a $+ + + +$ Wassermann test; a serum Wassermann test was not made. He failed rapidly, and died, June 7, 1918, one and one-half months after readmission, at the age of 37 years and 6 months.

CASE 11.—A driver, aged 36 years and 6 months, was admitted to the hospital, May 30, 1917. He had had syphilis at the age of 23. At the age of 36 years and 6 months, there began a gradual change of character, with irritability and forgetfulness. For one month before his admission to the hospital, he was elated and had expansive delusions of wealth and of his abilities. His orientation, memory and retention were defective. His pupils were irregular, unequal and reacted sluggishly to light. His knee reflexes were unequal and exaggerated. He had tremors of the tongue and facial muscles and slurring of the speech. After four months, he began to improve gradually. He was paroled, Sept. 22, 1917, after four months' residence in the hospital in "apparently a marked remission." He was considered normal by his family, and he worked steadily in his former position. He had "several intraspinal treatments." After twenty-three months, he suddenly became confused, and six days later, Aug. 16, 1919, he was readmitted to the hospital at the age of 38 years and 6 months. He was in a dull, restless state, and had defective orientation, memory, retention and calculation. His pupils were fixed. His knee reflexes were absent. The neurologic signs were otherwise unchanged. The cerebrospinal fluid gave a positive globulin reaction and contained twelve cells. The Wassermann test was $+ + + +$. A serum Wassermann test was $+$. Three months later, he had a series of convulsions. He died, June 22, 1920, ten months after readmission, at the age of 39 years and 6 months.

CASE 12.—A delicatessen storekeeper, aged 40 years and 6 months, was admitted to the hospital Dec. 13, 1916. He had had syphilis at an unknown date. Since the age of 40, he had gradually failed and had become restless, irritable and excitable; he talked of being wealthy and had had confused episodes. Seven months later, he was admitted to the hospital in an irritable, euphoric state; he

had an expansive trend, impaired orientation, memory and retention. His pupils were normal, his knee reflexes active. He had tremors of the tongue and fingers. The cerebrospinal fluid gave a positive globulin reaction and contained 227 cells. He had a + + + + Wassermann reaction. A serum Wassermann test was not made. His delusional trends had disappeared five months later. He appeared dull, and did not know the name of the hospital. "Mixed treatment was given." He was paroled, June 22, 1917, six months after admission, at the age of 41, in an improved condition. He remained at home for four months, then went to work keeping a stand and later as a pedler for two and one-half years. During this period, he received twelve intraspinal treatments of arsphenamin. He finally became confused, restless and incoherent. Six weeks later, April 3, 1920, he was readmitted to the hospital at the age of 43 years and 10 months. He was slightly elated and had marked orientation and memory defects. His pupils were irregular, small and sluggish to light. His knee reflexes were increased. He had tremors of the fingers and speech defect. The cerebrospinal fluid showed a + + globulin reaction and contained nineteen cells. He had a + + + + Wassermann test, and the serum Wassermann test was + + + +. He failed gradually, developed a series of convulsions, and died, May 30, 1920, at the age of 44 years, fifty-seven days after readmission.

CASE 13.—A salesman of electrical supplies, aged 32, was admitted to the hospital, Dec. 22, 1914. He had had syphilis at the age of 23. At the age of 31 he was out of work for nine months while the company was being reorganized. He was unable to return to work, was worried, excitable and abusive. He was admitted to a sanitarium, where he remained for three months. There he was resistive, assaultive and dull. On admission to the hospital at the age of 32, he was dull, irritable, resistive and at times mute. His pupils were small and sluggish. His knee reflexes were exaggerated. He had tremors of the tongue and fingers. The first examination of the cerebrospinal fluid revealed an increase of globulin and contained twenty-two cells. The result of the Wassermann test was not reported. In the second examination there was a positive globulin reaction and twenty-eight cells. The Wassermann test was + + + + and the serum Wassermann test was ±. He began to improve five months later, and was paroled Oct. 8, 1915, at the age of 33, as improved. He was idle for four months and then returned to his previous occupation. He had some insight, was talkative, exhibited slight emotional elevation and was careless with his money. He suddenly became noisy, restless and talked of having millions of dollars. Two weeks later, July 12, 1916, he was readmitted to another state hospital, where he died twenty-five days later, Aug. 6, 1916, at the age of 33 years and 6 months. The cerebrospinal fluid showed a + globulin test, and it contained eighteen cells. The Wassermann test was negative, as was also the serum Wassermann test.

CASE 14.—A dry goods salesman, aged 48, was admitted to the hospital, Dec. 12, 1911. He had had a chancre at the age of 25. At the age of 45, he had been refused life insurance because of unequal pupils. At the age of 46, he had had a nervous breakdown, during which he was nervous and forgetful and his mind was sluggish. He improved after six months. He had been impotent since. At the age of 47 years and 6 months, he had worked on the invention of a non-refillable bottle. He also had difficulty in finding the right word in talking. He suddenly became excited and had a feeling of exaltation. Two weeks later, he was admitted to the hospital in an inaccessible, noisy and restless excitement. His eyes were closed, and he grimaced and gesticulated. He had Argyll Robertson pupils. His knee reflexes were exaggerated. He had tremors of the tongue

and hands. The cerebrospinal fluid gave a positive globulin reaction and contained eighty-eight cells. The result of the Wassermann test on the fluid or serum was not reported. One month later, he showed marked improvement with insight and intact orientation and memory, although he was slightly euphoric. He was paroled, Feb. 13, 1912, one and one-half months after admission, as improved. After remaining one year at home, he secured, at the age of 49, a position as an insurance agent. At the age of 51 years and 6 months, he became "weak and run down." Finally, he had marked difficulty in walking, had a speech defect and convulsions and was irritable. On Aug. 15, 1917, at the age of 53 years and 6 months, he was readmitted to the hospital. He was dull and restless, had a mildly expansive trend, and defects of orientation, memory and writing. His speech was slurring. The cerebrospinal fluid gave a positive globulin reaction and contained seventy-one cells. The Wassermann test was $+ + + +$. The serum Wassermann test was $+ + + +$. Later he showed some improvement and was paroled for one and one-half months, after which he returned to the hospital. One year later, Nov. 15, 1918, at the age of 55, fifteen months after readmission, he died.

CASE 15.—An accident investigator, aged 52, was admitted to the hospital, July 10, 1913. He went on periodical sprees every two or three years. He had had syphilis at the age of 37. Since the age of 40, he had complained of neuralgia in the head and back. At the age of 51, a gradually progressive change was noted in his disposition, and he was easily excited and became irritable. One year later he became overactive, talkative, irritable and had an expansive trend about money. His orientation and memory were intact. He was unable to recall any retention tests. He had irregular Argyll Robertson pupils. His knee reflexes were diminished. He had tremors of the tongue and hands and slurring speech. The cerebrospinal fluid gave a positive globulin reaction and contained ninety-seven cells. The Wassermann tests were not performed. During his hospital residence of three months he became quieter, but there was a persistence of irritability. On Oct. 14, 1913, he was paroled as much improved. One month later he had a left hemiplegia which entirely cleared up without emotional instability, in two months. He returned to his former work for six months, when he had to stop because of incontinence of urine. Three months later, he again developed an acute psychomotor excitement with irritability, a boastful trend and a clear sensorium, except for varying errors in time relations of events of the recent past. His neurologic signs remained unchanged. The cerebrospinal fluid gave a positive globulin reaction and contained one cell. The Wassermann test was not performed. He was paroled, Nov. 25, 1914, after two and one-half months' hospital residence, at the age of 54. He went to work as a process server at which he has been steadily employed, earning $17.50 a week. Now at the age of 62 he appears well and is self-sustaining.

CASE 16.—A stableman, aged 40 years and 6 months, was admitted to the hospital, Dec. 3, 1914. He used alcohol in moderation. He married at the age of 35 and had one child. He denied having had syphilis. Four months before admission, he had a slight head injury. There were no orientation, memory or retention defects, nor errors in writing. He had no insight. His pupils were sluggish to light; the left knee reflex was diminished; the right was absent. He had tremors of the tongue. He had a speech defect. The cerebrospinal fluid gave a positive globulin reaction and contained fifty-four cells. The Wassermann test was $+ + + +$. A serum Wassermann test was not made. He improved in the hospital, had some insight and was paroled, Feb. 22, 1915, two and one-half months after admission. He remained at home one week and

received one intraspinal "treatment." He then returned to work. After three and one-half months, he became confused, irritable and indifferent. Within three weeks, he was readmitted to the hospital, July 23, 1915, at the age of 41. He was childish and euphoric, had good orientation and memory for outstanding events, but there were discrepancies in time relations of more recent events, and defects in retention. His pupils were irregular, and he had incoordination of the extremities. The neurologic signs were otherwise unchanged. There was little change in his condition for some months. In October, 1916, there was a rapid mental and physical decline, and he died, Dec. 18, 1916, seventeen months after readmission, at the age of 42 years and 6 months.

CASE 17.—A stationary engineer, aged 47, was admitted to the hospital, May 4, 1917. He had had syphilis at the age of 27. One month before admission, he had a complete change in disposition—he became obscene, irritable and expansive. In the hospital, he was dull and inactive. He had defects in orientation, memory and retention. He had irregular Argyll Robertson pupils. His knee reflexes were diminished. He had tremors of the tongue, facial muscles and fingers. He gradually improved, and was paroled, Nov. 15, 1917, six months after admission, at the age of 47 years and 6 months. He resumed his former occupation at once. His legs were tremulous; otherwise he appeared well. In June, 1918, for a period of three weeks, he was excitable and forgetful, after which he returned to his work, where he remained until September 1, eight and one-half months after parole, when he again became confused and irritable. Ten days later, Sept. 10, 1918, he was readmitted to the hospital. He was childish and slightly expansive and had marked defects of orientation, memory and retention. His knee reflexes were absent, and he had a speech defect. The neurologic signs were otherwise not changed. The cerebrospinal fluid gave a positive globulin reaction and contained seventy cells. The Wassermann test was + + + +. The result of a serum Wassermann test was not reported. He failed gradually, developed convulsions and died, March 30, 1919, six months after readmission, at the age of 50.

CASE 18.—A sculptor, aged 40, was admitted to the hospital, May 21, 1915. He had had syphilis at the age of 30. For two months, he had been restless and nervous and had exhibited a quick temper. For two weeks, he had expressed expansive trends of great wealth and personal ability. In the hospital, he was euphoric, overactive and expressed an expansive trend. His orientation, memory and retention were intact. His pupils were small and irregular, but reacted promptly. His knee reflexes were diminished. He had tremors of the tongue and facial muscles. The cerebrospinal fluid gave a positive globulin reaction and contained sixteen cells. The Wassermann test was positive. A serum Wassermann test was not made. He continued to be restless, unstable, expansive and assaultive. After nineteen months in the hospital, he became quiet and occupied himself. Six months later, after a hospital residence of twenty-five months, he was paroled, June 30, 1917, at the age of 42, as in a remission. Insight was lacking, but he showed no irritability. He went to work at wood carving, and earned $6.00 a day. He last reported, December, 1917, and was in the same condtion. He died suddenly in 1918 of a cerebral hemorrhage at the age of 43.

CASE 19.—A teamster and hostler, aged 51 years and 6 months, was admitted to the hospital, May 27, 1914. He had had syphilis at the age of 39. Nothing is known concerning the onset of his psychosis. He was picked up by the police in an acutely excited state, proclaiming that he was Jesus Christ, and was placed in a hospital in Salem, Ore., four weeks before his admission. On admission,

he was restless and euphoric, and claimed to be Jesus Christ, the savior of the world, and entertained other exalted delusions. His orientation was intact. His memory was good for gross events, but there were discrepancies in time relations. His retention was impaired, and there were errors in writing. His pupils were sluggish, and the knee reflexes were absent. He had tremors of the tongue, facial muscles and fingers. His speech was defective. The cerebrospinal fluid gave a positive globulin reaction and contained eight cells. The Wassermann test was positive. A serum Wassermann test was not made. He was paroled at the age of 52, after four months. For more than six months he remained at home. He then secured work as porter in a dry goods store. He appeared well, and worked efficiently. He suffered an injury of the head, lost considerable blood, became restless, confused and euphoric. Three months later, Sept. 18, 1920, at the age of 58, he was readmitted to the hospital. He was in a euphoric and expansive state, had persecutory delusions and was irritable. His orientation, memory, retention and calculation were defective. The neurologic signs were unchanged. The cerebrospinal fluid gave a + + globulin reaction and contained twelve cells. The Wassermann test was +. The serum Wassermann test was negative. He gradually failed, and died, March 21, 1921, at the age of 58 years and 6 months, after a hospital residence of six months.

CASE 20.—A chauffeur, aged 33, was admitted to the hospital, July 11, 1911. He had had syphilis at the age of 17. He had been nervous for years. On the day of his admission, he had talked of being worth millions. In the hospital he was elated, euphoric and expansive. His orientation and recent memory were good. His remote memory showed date discrepancies. He had some insight. His pupils were sluggish, his knee reflexes active. He had coarse tremors and a speech defect. At the first examination, the cerebrospinal fluid gave a positive globulin reaction and contained sixty-three cells. The Wassermann test was positive. The serum Wassermann test was negative. At the second examination, the cerebrospinal fluid gave a positive globulin reaction and contained eleven cells, and the Wassermann test was positive. The serum Wassermann test was also positive. Two months after admission, he was composed and had insight into his conduct and delusional trends. There were a few date discrepancies. He kept himself employed. After seventeen months of hospital residence, he was paroled, Dec. 7, 1912, at the age of 34 years and 6 months, as in a remission. He went to work and did well until shortly before his return to the hospital. He was readmitted, May 6, 1913, at the age of 35, after an extramural residence of five months. He was euphoric, somewhat expansive and had date discrepancies in the remote past, and defective retention. His orientation and recent memory were intact. Insight was absent. Aside from markedly diminished knee reflexes, there were no changes in the neurologic signs. He gradually became dull and developed memory defects, with confusion and convulsions. He died, April 18, 1916, at the age of 38, after a second hospital residence of three years.

CASE 21.—A plasterer, aged 36 years and 6 months, was admitted to the Central Islip State Hospital, June 3, 1913. He had had a chancre at the age of 19. Three weeks before admission, he had become irritable, restless and talkative, and expressed an expansive trend of wealth and personal abilities. His memory, judgment and insight were markedly defective. His pupils were unequal and sluggish. His knee reflexes were exaggerated. He had tremors of the tongue and fingers, slurring speech and swaying in the Romberg position. The cerebrospinal fluid had a marked lymphocytosis. After three weeks he began to improve and was paroled Sept. 21, 1913, after two months of hospital residence. Shortly after this, he had trouble with his wife and went to live with his brother.

He worked steadily, and his behavior was normal. After five months, he again had trouble with his wife and became depressed and fearful. He was readmitted to another state hospital, April 24, 1914, where he was dull, slow and had marked memory impairment. He gradually became destructive and uncleanly. He had an impaired sense of taste and smell, was tremulous and ataxic. The other neurologic signs were unchanged. The cerebrospinal fluid had a marked lympho-cytosis. The Wassermann test was strongly positive. After two months, he became quieter and was transferred to the Manhattan State Hospital, Nov. 17, 1914. Here he was indifferent, euphoric and expansive. He had a poor memory and marked speech defect. Six months later, he again became quiet and was paroled, June 24, 1915. Three weeks later, he was returned to the hospital in an expansive and disturbed state. He gradually failed, and twenty months after readmission developed convulsions and died Dec. 10, 1915, at the age of 39.

CASE 22.—A stenographer, aged 25, was admitted to the hospital, Feb. 21, 1917. He had had syphilis at the age of 20 or 21. No anamnesis was obtained. He had been arrested in the office of a prominent banking house where he had gone and announced in a noisy, excited manner "I am about one million better off than I was when you saw me." He was taken to the hospital where, on admission, he was overactive, noisy, excited and expressed a religious trend. He soon quieted down into a dull, inactive state, with defects of orientation and memory. His pupils were unequal; the left was fixed to light; the right was limited in reaction. His knee reflexes were exaggerated. He had tremors of the hands and fingers. The cerebrospinal fluid gave a positive globulin reaction and contained 138 cells. The Wassermann test was + + + +. A serum Wassermann test was not made. He was discharged, May 6, 1917, as unim-proved, as a nonresident to be returned to his home in Georgia. He remained under a physician's care until September, when he returned to work as a stenog-rapher. A report was received in May, 1923, five and one-half years after dis-charge, stating that he had been employed steadily, had not been nervous, and his memory was good. "Nothing has been observed to indicate he is not well in every way."

CASE 23.—A shoemaker, aged 35, was admitted to the hospital, April 16, 1914. He had had a chancre at the age of 23. For three or four weeks before admission, he had complained of distress in the region of his heart. For one week he had been uneasy, had slept poorly and was depressed and saw lights in the sky. In the hospital he was dull, sullen, excitable, suspicious and unable to tell what month or year it was. There were no memory defects. His pupils were unequal, the left immobile; the right reacted to light slightly. His knee reflexes were exaggerated. He had tremors of the tongue. The cerebrospinal fluid gave a positive globulin reaction and contained eighteen cells. The results of the Wassermann tests were not reported. He was paroled, Oct. 24, 1914, after six months' hospital residence, as improved. He returned to his work as a shoemaker, at which he continued for three years. Since then, for five and one-half years, he has worked as a porter in a club. He has been self-sustaining for eight and one-half years. Now, at the age of 44, he continues well.

CASE 24.—A laborer, aged 39 years and 6 months, was admitted to the hos-pital, Feb. 21, 1917. No anamnesis was obtained, and he denied syphilis. For an unknown period, he had been despondent and sat around the house. On admission to the hospital, he was dull, careless and inactive with, at first, depressed and excited episodes. He worried because he could not send money to his family in Europe (war). He had orientation and memory defects. His pupils were unequal and fixed to light. His knee reflexes were exaggerated. He

had tremors of the tongue, facial muscles and fingers and a speech defect. The cerebrospinal fluid gave a positive globulin reaction and contained fifty-six cells. The Wassermann test showed complete inhibition. A serum Wassermann test was not made. His general physical condition improved, although the neurologic signs persisted. He became more alert and had a better grasp on the situation. After four and one-half months, July 9, 1917, he was paroled as improved. He immediately went to work as a stevedore. At the expiration of six months, he was interviewed. He had continued at his work, felt well, appeared in good physical condition and was "quite alert to ordinary affairs." He soon returned to Austria. In 1922, at the age of 42 years and 6 months, a letter stated that he was doing well and working.

CASE 25.—A barber, aged 42, was admitted to the hospital, April 3, 1914. He had had syphilis at 18 and described secondary symptoms. At the age of 20, he had had dizzy spells and complained of not feeling right; and at 39, he had had difficulty in urinating. Since the age of 40, he had been nervous, depressed and weak. After having been unable to work for three months, he was admitted to the hospital, at the age of 42. He was dull and quiet and had difficulty in recalling past dates and in correlating time relations. He had unequal Argyll Robertson pupils. His knee reflexes were unequal; the right was exaggerated and the left was sluggish. He had tremors of the tongue and facial muscles. There was hyperesthesia from the umbilicus to the nipples. The cerebrospinal fluid gave a positive globulin reaction and contained ninety-seven cells. The Wassermann tests were not reported. He was paroled as improved, June 16, 1914, after three months in the hospital. He had Argyll Robertson pupils and absent knee reflexes. After one month, he went to work but changed positions frequently because of feeling tired and weak. For the past four years, since the age of 46, he has worked steadily selling barber supplies. The weakness in the legs did not entirely leave him until two years ago. At present, at the age of 51, his general physical condition is good. His speech is intact, and he walks well. There are no changes in his pupils. His memory is good for ordinary events. He feels well and is self-sustaining. He has received intravenous injections of arsphenamin at irregular intervals since his discharge.

CASE 26.—A silk jobber, aged 21, single, was admitted to a sanitarium, Jan. 26, 1906. No history of syphilis was ascertained. In the fall of 1905, his business had been ruined by a bad deal. He lost interest, became depressed, slow and inactive, and after four and one-half months was admitted to the sanitarium, where he passed successively through a retarded depressed state, an agitated state with ideas of impending death and poisoning, an overactive, talkative and mischievous state and a mute, inactive unclean, resistive state. After five months, May 22, 1906, he was transferred to the Manhattan State Hospital. His pupils reacted sluggishly to light. His knee reflexes were active. He had fine tremors of the tongue. A serologic examination was not made. For fourteen months he continued in the stuporous condition mentioned above. During the next ten months he was in an overactive, talkative, elated and abusive state of varying intensity, with good orientation and memory. He then began to improve, and after five months appeared to be recovered, with fairly good insight and good memory for his entire illness. He was paroled, Oct. 1, 1908, after two years and nine months' hospital residence, at the age of 23. The case was diagnosed as allied to manic-depressive psychosis. He went to work as a driver, and married. He became irritable, and in December, 1915, at the age of 31, began gradually to lose his memory, was easily excited, and four months later, April 11, 1916, he was readmitted to the hospital. He was dull, restless, bed-

ridden and unable to speak intelligibly. He had unequal and fixed pupils. His knee reflexes were absent. He had tremors of the tongue and facial muscles. A serologic examination was not made. He died six days later at the age of 31 years and 6 months.

CASE 27.—A cook, aged 49, was admitted to the hospital, Oct. 9, 1891. He had been intemperate. He was married but had no children. No history of syphilis was ascertained. At the age of 45, he had had a depression lasting two months, and at 47 he had had a depression lasting three months. At the age of 49, he was admitted to the hospital in a retarded depression, with defective memory and contracted pupils. He recovered after three months, and was discharged. The case was diagnosed as acute melancholia. He appeared well, and worked for four months. He then remained at home for sixteen months. Aug. 17, 1893, at the age of 51, he was readmitted in a mildly excited state. He had unequally contracted pupils, absent knee jerks and a tremulous tongue. After ten months, he was discharged as improved. The case was diagnosed as general paralysis. There was no interval history, and it is questionable whether he had been considered well. Ten years later, Aug. 20, 1903, he was readmitted at the age of 61. He had a depression with loss of memory and a mild persecutory trend. He had numbness of the left foot and leg, and his gait was unsteady. After thirteen months' hospital residence, he was discharged improved, with a diagnosis of chronic melancholia. Three months later, Dec. 3, 1904, he was readmitted at the age of 62. He had a retarded depression with memory impairment. His pupils were contracted and reacted sluggishly. His knee jerks were exaggerated and his tongue tremulous. Sept. 5, 1905, after nine months' hospital residence, he was paroled as recovered, with a diagnosis of a depressed type of manic-depressive psychosis. During a period of eight and one-half years he was considered well. He was readmitted, Feb. 14, 1914, at the age of 72, in an agitated depression of two months' duration. His orientation and memory showed few defects. He had unequal Arygll Robertson pupils. His knee jerks were unequally diminished. He had tremors of the tongue and fingers. The cerebrospinal fluid gave a positive globulin reaction and contained twenty cells. He rapidly failed, and died March 3, 1914, seventeen days after admission, at the age of 72. The case was diagnosed as one of general paralysis.

CASE 28.—A bookkeeper, aged 29 years and 6 months, was admitted to the Central Islip State Hospital, May 4, 1909. In 1902, at the age of 23, after his return from the Boer War he had been "nervous and restless and contracted syphilis." For a period of two and one-half years, since the age of 27, he had felt that his life was going away from him. For three months he had suffered from a feeling of insufficiency and depression with worry over his venereal infection. In the hospital, he improved rapidly. His orientation and memory were good. His retention was defective. His pupils reacted sluggishly; his knee jerks were diminished; he had tremors of the tongue and facial muscles and slurring speech. There was lymphocytosis of the cerebrospinal fluid. The patient was discharged after three months as "well." One month later, Oct. 7, 1909, he was readmitted. For four days, he had been overactive and euphoric, and had expressed delusions of wealth. His orientation and memory were intact. He had Argyll Robertson pupils. His knee reflexes were active, and he had tremors. After five months, he began to improve. He received two courses of inunctions. On May 24, 1910, at the age of 30 years and 6 months, after seven and one-half months' hospital residence, he was paroled as in a "remission in mental symptoms without any defects." During twenty months, he worked efficiently as a bookkeeper and received one increase in wages. On Jan. 11, 1912, at the age of 32

years and 6 months, he was readmitted. For eleven days he had been over-active, euphoric and had expressed expansive delusions. There was no impairment of orientation, but calculation was defective. His tremors were not marked, and his speech was slurring. His blood pressure was 190. There were no other changes in the neurologic signs. There was no gloubulin reaction of the cerebrospinal fluid, and it contained six cells. Both Wassermann tests were positive. Nine months later, there was a positive globulin reaction of the cerebrospinal fluid, and it contained twenty-two cells. The Wassermann tests were not reported. He gradually became more cleanly, but was tearful and had memory defects. One year after readmission, he was noted as improved; he worked in the ward; his memory and orientation were good; his retention was defective. He had insight into his previous condition. After six months' improvement, he again began to fail, and died, April 10, 1914, at the age of 35, two and one-fourth years after readmission.

CASE 29.—A fencing master, aged 37, was admitted to the hospital, June 8, 1912. He said that he had not had syphilis. For two years he had been "mentally upset," and had imagined that people were trying to poison him. In the hospital he was restless, simple in his manner, depressed and spoke of feeling depressed over business reverses. He had defects in orientation, memory and retention. His pupils were small but responded to illumination. He had exaggerated knee reflexes, tremors of the facial muscles and drawling speech. No serologic examination was made. After twenty-one months, he began to improve and worked in the store-house. After three years and ten months' hospital residence, he was paroled, April 6, 1916, at the age of 41. He worked for one year as "general helper." He then returned to his former vocation as fencing master. At present, at the age of 48, he appears well and has just completed a fencing exhibition engagement. He has been well and has maintained himself since leaving the hospital.

CASE 30.—A hoisting engineer, aged 45 years and 6 months, was admitted to the hospital, May 27, 1914. He had been intemperate. He had had syphilis at the age of 21. At the age of 42, he had fallen and had had a lacerated wound of his nose. During the ensuing two years and ten months, he continued to drink and was dull and forgetful. He had delusions of persecution, was jealous, and had auditory and visual hallucinations of varying intensity. He was abusive, threatening and assaultive. Following his admission, at the age of 45 years and 6 months, he was dull and irritable. His orientation was good. His memory was poor, with discrepancies in time relations. His retention was defective. He had Argyll Robertson pupils, exaggerated knee reflexes, tremors of the hands and swaying in the Romberg position. At the first examination, the cerebrospinal fluid contained thirteen cells. The globulin reaction was unascertained. The Wassermann test was negative. A serum Wassermann test was + + + +. At the second examination, the cerebrospinal fluid had a + globulin reaction, contained twenty cells, and the Wassermann test was positive. A serum Wassermann test was not made. After six months, he showed some improvement and was paroled, Oct. 19, 1914, at the age of 46. He remained at home, drank and was abusive. After four months, he returned to the hospital in the same condition as during his previous residence, but his speech was slurring. Two months later, April 20, 1915, at the age of 46 years and 6 months, he was again paroled as improved. He remained at home one year. He worked steadily as a packer in a wholesale house for three and one-half years. He appeared well but was more domineering and abusive than was natural to him. Nov. 9, 1920, at the age of 52, he had a convulsion with left hemiplegia and died.

CASE 31.—A stationary engineer, aged 34, was admitted to the hospital, July 23, 1916. He had had a chancre at the age of 20. He had been intemperate and had had delirium tremens three months before admission. He had been drinking for two weeks before admission. He complained of headaches and noises in his ears and held his fingers in his ears. On admission to the hospital, he was fearful because of auditory hallucinations and delusions of persecution. He was poorly accessible to questioning, was dull and made impulsive assaults. He thought something was wrong with his mind. His orientation was poor for time, but his memory was fair. His retention was poor. He calculated fairly well. He had irregular pupils which reacted promptly to light. His knee reflexes were diminished. He had coarse tremors of the tongue, facial muscles, hands and fingers. He had a washed-out appearance of the face. His speech was slurring. At the first examination, the cerebrospinal fluid gave a positive globulin reaction and contained thirty-four cells. The Wassermann test was not performed. A serum Wassermann test was positive. At the second examination, the cerebrospinal fluid gave a positive reaction and contained fifty-six cells. Both fluid and serum Wassermann tests were + + + +. During a period of ten months at the hospital, his condition fluctuated from being fairly composed, dull and free from delusions to a condition of many delusions and hallucination of all the senses with an antagonistic reaction to them. He was transferred to a private sanitarium and was discharged, Oct. 27, 1917, at the age of 35, as improved, after a total hospital residence of eleven months. An inquiry in July, 1923, developed that he had resumed his former occupation shortly after discharge. He is looked on as being very peculiar. He has worked irregularly because he does not find a job that suits him; he is rather irritable, but he is self-supporting.

CASE 32.—A shoemaker, aged 46, was admitted to the hospital, Feb. 25, 1916. He said that he had not had syphilis. On Jan. 1, 1916, he accused his wife of infidelity. He went to New York from Chicago for a rest. He had slept poorly, was nervous, absent-minded, careless in appearance and depressed. He had periods when he was anxious and fearful. He thought that he would be sent out of the hospital and killed by a man who desired his wife. His orientation and memory were intact. His retention was poor. He had unequal, irregular pupils, which accommodated themselves and reacted to light promptly. His right knee reflex was overactive, his left knee reflex diminished. He had fine tremors of the tongue and fingers. There was a slight hesitancy in his speech. The cerebrospinal fluid gave a positive globulin reaction and contained seventeen cells. The Wassermann test was not made. A serum Wassermann test was positive. He was discharged, Feb. 28, 1916, three days later, as unimproved. From an inquiry, May 19, 1923, it was learned that following his return home he spent a few weeks on his brother's farm. He then returned home and to work at his trade, at which he has been engaged since. At present, at the age of 53, he is "well in every way."

CASE 33.—An actor, aged 31, was admitted to the hospital, March 9, 1911. He had had syphilis at the age of 21. At the age of 30 years and 6 months, he had become childish, unstable, fretful and thought people had turned against him. His orientation and memory were good. He had slight defects of retention. He had Argyll Robertson pupils, absent knee reflexes, unsteady gait, ataxia and tremors of the tongue and facial muscles. The cerebrospinal fluid gave a negative globulin reaction and contained no cells. Both the fluid and serum Wassermann tests were positive. After three months in the hospital, he was paroled, with insight and as in a "remission." During the next four years, he worked

most of the time as advance agent for his actor brother and as a street car conductor and a subway guard. At the age of 35, he became depressed, worried, slept poorly, slapped himself and talked much about his inability to support his family. Two weeks later, Sept. 25, 1916, he was readmitted to the hospital. He had good orientation and recent memory. His remote memory showed defects. He was unable to correlate time relations, and he omitted letters in written words. The neurologic signs had progressed, and he showed sensory changes on the arms, abdomen and legs. The Wassermann tests were again both positive. On Jan. 3, 1916, three and one-half months later, he was paroled in an improved mental condition. During the ensuing four years, he was in the hospital for short periods of time on four occasions. On April 11, 1920, at the age of 40, he left the hospital in an improved condition. He was alert, had a good grasp on his surroundings and understanding of his condition. Since then he has maintained himself, although he was worked irregularly. He has had six intraspinal and eighteen intravenous arsphenamin treatments. At present, at the age of 43, he is emotionally elevated and unstable. He has slight intellectual defects, but is able to work, selling bottled soda. His neurologic signs showed a marked increase in ataxia and a tabetic gait and tremors of the tongue and facial muscles.

COMMENT ON CASES REPORTED

In Case 4, the patient had a psychosis characterized by a dull, drowsy state of four years' duration, followed by a "well" interval of six years before the onset of the psychosis definitely diagnosed as general paralysis.

In Case 14, the patient was found, at the time of a life insurance examination, to have unequal pupils, although the mental symptoms were not in evidence until one year later.

In Case 15, the patient had a history of "neuralgia" in the head and back for twelve years before the onset of the mental symptoms; also two periods of remission of six months and eight years, respectively. He is still living.

In Case 27, the patient had, beginning at the age of 45, six depressed and one excited episodes. In the third episode, and during the first admission, he was noted as having contracted pupils. In the fourth episode and second admission, at the age of 51, he had equally contracted pupils, absent knee reflexes and tremulous tongue, and his condition was diagnosed as general paralysis. The physical signs continued throughout subsequent residences, during which the case was looked on as one of manic-depressive psychosis, until his last admission, at 72, when his condition was again diagnosed as general paralysis.

In Case 31, the patient was intemperate, and alcohol undoubtedly played a part in his hallucinatory and delusional state.

In Case 33, the patient was not readily classified in the four clinical groups. The onset showed changes in disposition with emotional instability and paranoid trend, followed by a period of remission of four years, which in turn was followed by a depressive reaction and then a remission of three years plus. The patient is still living.

The neurologic signs in these cases presented no noteworthy variations. Twenty-three patients presented increased knee reflexes; Cases 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 20, 21, 22, 23, 24, 26, 29, 30, 32. Nine patients presented diminished or absent knee reflexes; Cases 15, 16, 17, 18, 19, 27, 28, 31, 33. One patient, Case 25, presented one exaggerated and one sluggish knee reflex.

TABLE 3.—*Age Distribution of Remissions by Clinical Groups*

Age	Clinical Groups					Total
	1	2	3	4	5	
21	—	—	1	—	—	1
25	—	1	—	—	—	1
29½	—	—	1	—	—	1
31	—	—	—	—	1	1
32	1	—	—	—	—	1
33	—	1	—	—	—	1
34	1	—	—	1	—	2
35	—	—	1	—	—	1
36½	1	1	—	—	—	2
37	1	—	—	1	—	2
38	1	—	—	—	—	1
38½	1	—	—	—	—	1
39½	—	—	1	—	—	1
40	—	1	—	—	—	1
40½	2	—	—	—	—	2
41½	1	—	—	—	—	1
42	—	—	1	—	—	1
43	1	—	—	—	—	1
44	1	—	—	—	—	1
45	1	—	—	—	—	1
45½	—	—	—	1	—	1
46	—	—	—	1	—	1
47	1	—	—	—	—	1
48	1	—	—	—	—	1
49	—	—	1	—	—	1
51½	—	1	—	—	—	1
52	2	—	—	—	—	2
53	1	—	—	—	—	1
Total	17	5	6	4	1	33

Table 3, indicating the age distribution at the time of admission by clinical groups, shows that of thirty-three patients with remissions, sixteen were admitted before the age of 40 and seventeen after that age. Group 1, the largest of the groups, shows a wide variation in age distribution.

TABLE 4.—*Clinical Group Distribution of Cases with Remissions*

	Group 1	Group 2	Group 3	Group 4	Group 5	Total
Dead	11	4	3	1	—	19
Living	6	1	3	3	1	14
Total	17	5	6	4	1	33

Table 4 shows the group distribution and whether the patients are now living or dead. Group 1 constitutes one-half—seventeen of the thirty-three cases; Groups 2, 3 and 4 are about evenly divided; Group 5, one case. In the seventeen cases in Group 1, eleven are dead and six living; in Group 2, four are dead and one is living. This is of

interest as it has been held by some that this type of case offered a better prognosis for spontaneous remissions. Of the thirty-three cases, more than one-half—nineteen—are dead and fourteen are living.

Table 5 shows the group distribution according to the duration of the psychosis before admission. Of the seventeen Group 1 cases, more than one-half—twelve—had a duration of one year or more; of the three

TABLE 5.—*Clinical Groups According to Duration Before Admission*

Duration	Group 1	Group 2	Group 3	Group 4	Group 5	Total
Less than 1 month	—	1	—	1	—	2
1 month	1	1	1	—	—	3
2 months	—	1	—	1	—	2
3 months	1	—	—	—	—	1
4 months	1	—	—	—	—	1
6 months	2	—	1	—	1	4
1 year	4	—	—	—	—	4
2 years	3	—	1	1	—	5
2½ years	—	—	1	—	—	1
3 years	1	—	—	1	—	2
4 years	3	—	1	—	—	4
10 years	1	—	—	—	—	1
Unascertained	—	2	1	—	—	3
Total	17	5	6	4	1	33

TABLE 6.—*Duration of Period of Remissions*

Duration of Remission	Living		Dead		Total	
	Patients	Remissions	Patients	Remissions	Patients	Remissions
3½ to 4 months	—	—	1	2 (1, #27)	1	2
5 months	—	—	3	3	3	3
6 months	—	1 (#15)	3	3	3	4
7 months	—	—	1	1	1	1
8 months	—	—	1	1	1	1
9 months	—	—	1	1	1	1
1 year	—	—	—	1 (#14)	—	1
1½ years	1	1	1	1 (#14)	2	2
1 year 7½ months	—	—	1	1	1	1
1 year 11 months	—	—	1	1	1	1
2 years	—	—	1	1	1	1
3 years	—	1 (#33)	—	—	—	1
3½ years	—	—	1	1	1	1
4 years	2	2 (1, #33)	—	—	2	2
4½ years	1	1	1	1	2	2
5 years	1	1	—	—	1	1
5½ years	1	1	1	1	2	2
6 years	2	3 (1, #4)	—	—	2	3
7 years	1	1	—	—	1	1
8 Years	3	3 (2,#15&4)	1	1	4	4
8½ years	1	1	1	1 (#27)	2	2
10 years	1	1	—	—	1	1
Total patients	14	—	19	—	33	—
Total remissions	—	17	—	21	—	38

Figures in parenthesis indicate how many cases are referred to and the number of the individual case.

ascertained cases of Group 2, none had a duration of more than two months; of the thirty-three patients with remissions, thirteen had a duration of less than one year, seventeen of one year or more, and three were unascertained.

Table 6 gives the duration of the remissions according to the present information. It will be noted that the thirty-three patients had thirty-

eight remissions. In Case 4, the patient had two remissions of six years and more than eight years, respectively. This patient is still living. In Case 14, the patient had two remissions of one year and one and one-half years; in Case 15, the patient had two remissions of six months and more than eight years (living); in Case 27, two remissions of four months and eight and one-half years; and in Case 33, two remissions of four years and more than three years, and is still alive.

Of the thirty-three patients, twelve had remissions of one and one-half years or less—two of these patients had second remissions of eight years and eight and one-half years; sixteen had remissions of three and one-half years or less—one had two remissions of one year and one

TABLE 7.—Race Distribution

Race	Total		Died During First Admission		De- ported No Data	Living in Hos- pital	Paroled, Re- turned and Died	Discharged		Remission	
	Num- ber	Per Cent.	Num- ber	Per Cent.				Unim- proved	Im- proved	Num- ber	Per Cent.
African....	45	4.4	36	80	4	1	1	1	2	—	—
Armenian..	5	—	4	—	1	—	—	—	—	—	—
Chinese....	5	—	4	—	1	—	—	—	—	—	—
Dutch......	6	—	5	—	1	—	—	—	—	—	—
English....	32	3.1	21	65	3	1	1	1	4	2	6.0
Finnish....	1	—	1	—	—	—	—	—	—	—	—
French.....	10	0.9	7	70	2	—	—	—	—	1	10.0
German....	146	14.5	118	80	8	4	2	6	5	3	2.0
Greek......	6	—	5	—	1	—	—	—	—	—	—
Hebrew....	229	22.8	186	80	6	2	4	10	14	7	3.0
Irish.......	155	15.4	123	79	7	8	2	5	4	6	3.8
Italian.....	97	9.6	76	78	7	2	4	3	3	2	2.0
Japanese...	1	—	—	—	1	—	—	—	—	—	—
Magyar....	18	1.7	16	—	1	—	1	—	—	—	—
Portugese..	1	—	1	—	—	—	—	—	—	—	—
Scandinavian	14	1.3	9	64	1	—	—	2	1	1	7.0
Scotch.....	11	1.0	10	90	—	—	—	—	—	1	9.0
Slavonic...	29	2.8	22	75	4	—	2	—	—	1	3.3
Spanish....	6	—	4	—	2	—	—	—	—	—	—
Syrian.....	1	—	1	—	—	—	—	—	—	—	—
Turkish....	1	—	1	—	—	—	—	—	—	—	—
Welsh......	1	—	1	—	—	—	—	—	—	—	—
Mixed......	138	13.7	98	73	11	2	3	8	11	5	3.5
Unascertained	46	4.5	35	76	1	1	1	—	4	4	8.0
Total....	1,004	—	784	78	62	21	20	36	48	33	3.5

and one-half years; and seventeen had remissions of four years or more; one had two remissions of three years and four years; one of six and one of eight years, respectively.

Of the fourteen patients now living, one had a remission of one and one-half years and thirteen of four years or more—one had two remissions of six months and eight years; and eight had remissions of not less than six years—one had two remissions of six months and eight years.

Of the patients who died, ten had remissions of less than one year; five had remissions of less than four years—one had two remissions of one and one and one-half years, respectively; and only two had remissions of more than six years, eight and eight and one-half years,

respectively. The latter had also had a previous remission of four months.

Table 7 gives the race distribution for the entire group as well as for the subdivisions of the group as shown in Table 1. The Hebrew, Irish and German races constitute more than half of the series. The African, 4.4 per cent., was the only race constituting 2 per cent. or more of the total which did not have remissions. The rates of remissions for each of the five largest race groups were as follows: Hebrew, 3 per cent.; Irish, 3.8 per cent.; Germans, 2 per cent.; mixed, 3.5 per cent.; Italian, 2 per cent. The Irish and mixed races were, therefore, the only large groups in which the remission rate of the race equaled the rate for the entire group, i. e., 3.5 per cent.

It is felt that the size of the group studied is large enough to draw fairly trustworthy conclusions as to the course of general paralysis in untreated cases. The length of time elapsed since admission and since leaving the hospital has been sufficiently long to warrant the assumption of a true remission of clinical symptoms in those patients who have been able to meet the requirements laid down as criteria of a remission.

SUMMARY

This study comprises 1,004 consecutively admitted male general paralytic patients with 1,043 admissions.

There were 784 first admissions, or 78 per cent., who died at the end of a continuous hospital residence. A total of 882 patients, or 87.8 per cent., are known to have died.

Of twenty patients, there are no data subsequent to their discharge from parole.

Forty patients are still living.

One hundred and thirty-eight patients, or 14.6 per cent., left the hospital.

Eighty-five patients, or 9 per cent., left the hospital improved.

Thirty-three patients, or 3.5 per cent., had true remissions of the clinical symptoms.

In more than one half of the patients with remissions, the duration of the psychosis before admission was one year or more.

Approximately one third of the patients had remissions lasting one and one and one-half years or less; one-half had remissions lasting three and one-half years or less; and one-half had remissions lasting four years or more.

The Hebrew race constituted the largest race group, i. e., 22.8 per cent. of the total.

The Hebrew, Irish and German races constituted more than one-half of the entire group, i. e., 52.7 per cent.

The African was the largest race group having no remissions—forty-five cases, or 4.4 per cent.

Of the four largest groups, Hebrews, Irish, Germans and mixed races, the Irish and mixed races were the only groups in which the rate of remissions was equal to or larger than the rate of the total group.

CONCLUSIONS

From this study it may be concluded that spontaneous remissions in patients with untreated cases of general paralysis occur but are not frequent; that in at least more than one half of the cases they are not permanent; that remissions may occur more than once in the course of the disease in the same persons; that remissions are more common in cases presenting a gradual onset, with changes in the disposition, emotional instability and defects in orientation and memory than in other clinical types of general paralysis; that there are no anamnestic, mental or neurologic criteria on which a prognosis for a remission can be based; that factors favorable to the occurrence of spontaneous remissions must be sought elsewhere than in the clinical picture of the disease; that the Jews, Irish and Germans are relatively more prone to develop general paralysis than other races; that the incidence of spontaneous remissions in Jews, Germans and Italians is low as compared with that of other races.

Kings Park, N. Y.

NEUROPSYCHIATRIC SEQUELAE OF CEREBRAL TRAUMA IN CHILDREN *

EDWARD A. STRECKER, M.D.

AND

FRANKLIN G. EBAUGH, M.D.

PHILADELPHIA

In reviewing the literature on head injuries, we were struck by the attention devoted to the acute manifestations of cerebral trauma [1] and the rarity of observations concerning the late complications. The serious deferred results of head injury in adults, as discussed by Michael,[2] are even more pronounced in children. Here they attain vital clinical and social significance, since the physical, neurologic, psychiatric, and psychologic findings bear a close relation to school progress, delinquency, criminality and future adjustability in general. During the past year, thirty children with histories of previous head injury have been referred to us. There were included twenty-three boys and seven girls, ranging in age from 3 to 16. The intervals between the date of head injury and time of first examination were: in seven, four months to one year; in four, one to two years; in seven, two to five years; in seven, five to ten years; in five, more than ten years.

In each case, we considered the previous personality and adjustment of the child, the immediate symptoms, treatment following injury, type of trauma and the chronological development of the characteristic physical and psychiatric syndromes.

PHYSICAL SEQUELAE

The physical findings can best be considered under: (1) immediate results of injury; (2) type of cerebral damage—concussion, fracture, etc.; (3) subjective complaints following the injury.

The immediate results following the head injury, according to the histories obtained, showed that twenty-three of our patients had cerebral

1. Frazier, C. H., and Ingham, S. D.: Review of Effects of Gunshot Wounds of Head Based on Two Hundred Cases at U. S. General Hospital No. 11, Arch. Neurol. & Psychiat. 3:17 (Jan.) 1920. Wilensky, A. O.: Neurologic Manifestations of Fracture of Skull, Surg. Clin. N. Am. 1:1709 (Dec.) 1921. Wilson, A. K.: Role of Trauma in Etiology of Organic and Functional Nervous Disease, J. Neurol. & Psychopath. 4:133-147 (Aug.) 1923.

2. Michael, J. C.: The Old Head Injury Case, J. A. M. A. 80:1047 (April 14) 1923.

concussion with characteristic loss of consciousness, headaches and vary-
ing degrees of shock. Roentgenograms in six cases revealed definite
fractures. One boy (Case 23) had traumatic rupture of both ear drums.
In two instances, the progressive stupor together with other evidence of
increasing intracranial pressure demanded early decompression. One
boy gave clear evidence of intracerebral hemorrhage.

Headache was the common subjective complaint and occurred in
80 per cent. of the entire group. Often it was referred to the site of
the head trauma. Occasionally it was neuralgic in type, but in the
majority of cases it was diffuse and dull.

Vertigo appeared in approximately one-third of the series. The
headache and vertigo in many children seemed to be related definitely
to physical activity. Vertigo could be produced in several by sudden
changes in posture. It was worth noting that many parents and teachers
spoke of the marked fatigability of these children in the face of pro-
longed physical or mental exertion. Insomnia, although not constant,
was often noted. Extreme sensitiveness to ordinary noises was usual,
and here may be traced a connection with mental overactivity and
affective disorders. Several complained of feeling worse in a warm
room. It can readily be appreciated how these physical syndromes led
to maladjustment at school as well as to later industrial handicaps.

The neurologic findings may be conveniently grouped under the
following headings:

1. *Olfactory Sense.*—No involvement.

2. *The Eyes.*—Small, unequal, or sluggish pupils were observed in
three cases. Sluggish reaction to light was marked in two cases.
Paralysis of the external rectus muscle was noted in three cases,
nystagmoid movements in two cases. The visual fields were not charted.
Ophthalmoscopically, one patient showed optic atrophy.

3. *Anterior Hind-Brain Segment.*—This was not involved in any
case.

4. *Middle Hind-Brain Segment.*—The abducens function has already
been noted. Paralysis of the facial nerve was found in four cases.
Taste was normal. Deafness was present in two cases.

5. *Posterior Hind-Brain Segment.*—Taste was normal in all cases.
Negative findings were obtained for cranial nerves of this group.

6. *Cord Segments.*—These were normal. No loss of sensibility was
noted in the entire group. Evidence of pyramidal tract involvement
was found in two cases with hemiplegia, overactive reflexes, clonus,
and Babinski sign.

7. *Vasomotor Disorders.*—These were present in marked degree in
ten cases. There was flushing, pallor, marked intolerance to heat and
excessive sweating.

8. *Motility Defects.*—Ataxia was present in two cases, tremor of the hands in three.

9. *Convulsions.*—These occurred in five children and were of the typical generalized epileptic type. Epileptic equivalent states were marked in two cases. One patient gave an unverified history of jacksonian seizures.

POST-TRAUMATIC BEHAVIOR DISORDERS

Since most of the cases were not seen immediately following the injury but were referred after prolonged manifestation of abnormal mental reactions, the direct traumatic deliria do not enter into consideration. We are, therefore, concerned with (1) the traumatic constitution and (2) the traumatic defect conditions. Fifteen cases were found under each category.

Traumatic Constitution.—In these children there was often a history of explosive outbreaks in school or at home, frequently of such severity as to make the child a menace to its associates. Several children made threats to kill. One boy (Case 7) threatened to kill his mother and frequently struck her. In Case 14, there was a total change in behavior reactions. The boy was uncontrollable, destructive, and at times had wild temper tantrums during which he destroyed furniture. He enjoyed pinching and scratching his schoolmates, and tortured animals. The overactivity in these cases was definitely related to an impure affect (tension state). In some patients, as in Case 2, the mental reaction simulated a manic state with great psychomotor drive. This boy, aged 7, was also sexually precocious. This entire group of patients were unmanageable in school, and many had had extensive court experiences. The following instance is fairly typical.

H. M., aged 13, was referred to the clinic, May 1, 1923. At the age of 3, the child had fallen from a second story window striking on his head. Following this injury the eyes became crossed and the boy complained frequently of headaches. Behavior disorders appeared at the age of 5. There were frequent impulsive outbreaks with destructiveness and violence. Once he struck his foster father in the abdomen, causing serious internal injury, which necessitated immediate hospital care. The boy was emotional, jumping from one extreme to another, showing at one time great affability and at another extreme irritability. There were also definite spells of depression. Then he declared that life was not worth while, and he was potentially suicidal. The roentgenographic findings, neurologic examination, and all special examinations gave negative results. The intelligence quotient was 92.

This patient had been studied in several clinics. In one, the episodic outbreaks were considered as manic reactions. In another, the case was considered one of dementia praecox. There has been no improvement during our observation, and only the unwillingness of relatives and social organizations has kept him from being committed to an institution.

This case is of interest in that it shows a behavior disorder with trend reactions of a depressive as well as paranoid nature. The chronologic sequence of events refers the present condition to the severe head injury received at the age of 3.

The relationship to delinquency is important in these cases. For instance, the patient in Case 18 lied and stole extensively. In Case 24, a boy aged 13, forged checks for large amounts. The delinquency is probably linked with the fatigability and attention defect.

Traumatic Defect.—Evidence of behavior disorders in the cases of traumatic defect conditions were observed in fifteen of the series. Of these, the clinical nature of the underlying organic defect was revealed by amnesias, periods of confusion and epileptoid states, and frequently there was evidence of deterioration and mental deficiency (observed in six cases). Whether or not the mental deficiency was caused by the head injury, cannot be proved in each instance. However, it is certain that cerebral trauma was followed by marked retardation in typical cases. In Case 9, for instance, following the injury at 11 months there was marked regression in the previously acquired ability to walk and talk. As in the traumatic constitution group, definite neurologic findings were the rule. Two had areas of bone defect resulting from a decompression. Several showed typical traumatic epilepsy in addition to the findings summarized under the heading of the neurologic examination. Patients A. H and R. C. are illustrative.

A. H., a boy, aged 10, was referred to the clinic, April 12, 1923, because he ran away from home and did not progress in school. There was a history of cranial injury three years previously. He had been struck over the head with a heavy broom, and a few hours later complained of headache and drowsiness. He was unconscious for thirty-six hours. From this time, he progressively deteriorated in school work, had memory lapses for prolonged periods and was confused and overactive. On several occasions, he wandered about the city aimlessly for days. Once he was found riding back and forth in the subway in a confused mental condition. At home, he was emotional and irritable, and complained of headaches and dizziness.

He was admitted to the psychopathic ward for observation and appeared confused, but cooperated fairly well in the ward routine, developed interest in his surroundings and showed no amnesic periods. However, he could not state the length of time that had elapsed since his admission, and retention, general information, and calculation were extremely poor. After two weeks he escaped from the hospital grounds and was not located until two days later, when he was found wandering around in a totally different section of the city unable to tell what had happened in the interval.

R. C., a boy aged 12 years, was admitted to the Pennsylvania Hospital, June 19, 1919. Two years before he had been struck by a taxicab, and his skull was fractured. The history shows that he was normal before receiving the injury. After the accident, he began to have epileptic seizures, which have recurred at irregular intervals since. He has not progressed in school, frequently runs away from home and does not respond to discipline; during the past three years he

has grown constantly worse. The school authorities thought he was an imbecile. Roentgen-ray examination revealed a fracture in the occipital region extending from the left side of the occipital protuberance to the right mastoid process. The final diagnosis was traumatic epilepsy with mental deficiency, and the patient was recommended for admission to an institution for mental defectives.

Several other cases of this type are included in the group. The failure to progress in school is particularly marked, and there is progressive loss of mental capacity and ability, leading ultimately to total inability for intellectual training. Early admission to institutions for defectives where training along manual lines is given and close supervision is possible should be a more frequent recommendation.

PSYCHOLOGIC FINDINGS

From the standpoint of experimental psychology, fatigability seemed to deserve particular attention. It was noted in giving the usual psychometric examination that fatigue was always demonstrable. By

TABLE 1.—*Auditovocal Memory Span in Philadelphia General Hospital Group*

Name	Terman Binet Simon Intelligence Quotient	Auditovocal Memory Span for Digits	Number of Repetitions Required to Reproduce Next Higher Span	Average of Repetitions Required by Normal Children of Same Chronological Age Who Were Similarly Tested
I. B.	79	5	8	3.2
R. C.	76	5	8	3.2
R. C.	58	4	15	3.6
A. H.	97	8	2	3.6
G. M.	81	6	8	3.2
H. N.	82	6	8	3.6
N. S.	...	...	...	...
C. T.	77	6	5	3.6
H. T.	91	7	6	3.6
H. V.	66	7	9	3.4

fatigue we mean "a condition caused by activity, in which the output produced by activity tends to be relatively poor; and the degree of fatigue tends to vary directly with the poorness of the output." No satisfactory performance test has been evolved [3] to measure such fatigue. The immediate memory test with auditovocal memory span for digits [4] was the test used in this examination. After the span was established, the next highest span was tried, and the number of needed repetitions noted. It was observed that in traumatic cases a greater number of repetitions were required. For instance, H. N. had an auditovocal memory span for six digits and required eight repetitions in order to get to seven, while W. D., a normal child having the same span and

---

3. Muscio, B.: Is a Fatigue Test Possible? Brit. J. Psychol. **12**:31-46 (June) 1921.

4. Humpstone, H. J.: Some Aspects of the Memory Span Test. Study in Associability, Psychiat. Clin. Press, 1917.

Table 2.—Summary of Cases

Name	Age	Sex	Date and Type of Injury	Neuropsychiatric Sequelae	Roentgen-Ray Findings	Diagnosis	Present Condition
1. J. A.	5	M	Fell at 18 months; unconscious for ½ hour; discharge from right ear	Unable to walk for 1 year; now ataxic; indistinct speech; marked irritability; right facial weakness; easily excited, trembles, restless	....	Mental deficiency; traumatic defect	Unimproved; unable to progress in school; attending speech clinic
2. I. B.	7	M	Fell from fence when 6 years old	Hyperkinetic, sexually precocious, manic-like outbreaks; occasional epileptic convulsions; homicidal; no neurologic findings	Negative.....	Traumatic defect	Unimproved; institutionalized
3. I. B.	12	M	Onset on way to America; was struck on head by a sailor; delirious period followed	Extremely mischievous, impractical, contrary, with periods of excitement and sleeplessness; violent temper; noisy and unmanageable	....	Traumatic constitution; epilepsy	Unknown; child was deported
4. I. B.	6	M	Struck by street car when 3 years old; dragged under car for two blocks	Definite convergence strabismus; chronic spasms of arms; head turned but does not lose consciousness; attacks last a few minutes, from 1 to 2 weeks apart; swears, uses vulgar language with destructive and unmanageable periods	....	Traumatic defect with epilepsy	February, 1924: No more spells; continues to have temper outbreaks and destructive tendency; in second grade in school; progress in school fairly normal
5. A. C.	6	F	Fell out of bed when 7 mos. old; struck head, particularly left side, forehead	Did not develop normally after fall; never talked normally; backward in walking; fell frequently until 4 years old	"The convolution markings all over the inside of this skull are exaggerated; frontal sinuses and mastoids underdeveloped; the sphenoidal sinus and sella turcica are normal; sutures are, perhaps, a little abnormally distinct; believe child is suffering from some form of intracerebral pressure."	Traumatic defect	Attending school for deaf; improving
6. R. C.	12	M	Struck by taxi when 10 years old; skull fractured; in hospital from June 19, 1919, to July 10, 1919	Always normal before accident; spells began 2 months afterward and have been occurring at irregular intervals since; runs away and resents discipline	A linear fracture beginning in the occipital protuberance on left side and extending around the right side to upper border of the mastoid process	Traumatic epilepsy; mentally deficient	Unimproved; recommended for admission to institution for feebleminded
7. S. D.	12	M	Fell down cellar steps when 5 years old, striking head	After fall began to stutter; very excitable; reported in school as using abusive language; threatened to kill mother many times	....	Traumatic constitution	Stutters badly; memory span very poor; inattentive; gradual improvement
8. L. D.	12	M	Struck by truck in 1922; brought to hospital unconscious	Poor school progress; at age of 13 years reached the third grade; now working in factory	No evidence of bone injury to the skull; "I believe, however, there is distinct evidence of intraventricular pressure; I suspect this child will be found mentally defective"	Traumatic defect; mental deficiency with hydrocephalus	Unimproved
9. H. D.	11	F	Fell, when 11 months old, from high-chair, striking back of head	Showed some evidence of learning to walk, which did not continue after fall; unable to walk until 3 years old; speech defective up to present time; left leg shorter; incoordination left hand; intelligence quotient 41.4	Negative.....	Traumatic defect; mental deficiency	

No.				History	Clinical notes	Fracture of the outer table of the skull from frontal eminence backward to above left ear	Diagnosis	Outcome
10.	J. D.	9	M	Aug. 29, 1920, struck by automobile, unconscious 3 days	Mental changes; very indifferent, apathetic; can be aroused only by shouting his name loudly several times; never able to give account of his accident; internal strabismus right eye, optic disk pale; pupils react; scars on both frontal bosses, more marked on left; incontinent urine and feces; right ear drum perforated but both bone and air conduction good in both ears, better in right; gait and coordination normal; mental development normal for age; active during interview; child frequently picked up by police and well known in court and House of Detention; begs on streets; series of arrests; no progress in special class		Traumatic constitution	Unimproved
11.	S. E.	8	M	When 1½ years old, fell out of bed, striking head; vomiting, stentorous breathing, stuporous condition; rapid pulse; overactive reflexes; suggestive Babinski sign; ankle clonus	Seemed unable to see after accident; difficulty in moving legs; apparent loss of power in right leg; restless, irritable; cried when touched; unable to concentrate; resists all restraint or discipline; 407 cells in spinal fluid; Wassermann test negative	Signs of concussion; no demonstrable fracture	Concussion of brain; intracerebral hemorrhage; mental retardation	Unimproved
12.	H. G.	6	M	Struck by automobile and brought to hospital Nov. 12, 1923, unconscious; bleeding from forehead; subtemporal decompression performed Nov. 14, 1923	Right facial weakness; overactive reflexes; doubtful Babinski sign; marked cervical rigidity; no true Kernig's sign; laboratory tests all negative	....	Traumatic constitution	Not known
13.	A. H.	10	M	Three years ago, struck over head with heavy broom; few hours later complained of headache, became very ill and was unconscious for 36 hours	Unable to progress in school; received 41 as general average; incorrigible; possible memory lapses at times; appeared confused, overactive; frequently complained of headaches and dizziness; possible amnesic periods; runs away; emotional; irritable	No demonstrable evidence of fracture either recent or old	Traumatic defect	Kept for observation in psychopathic ward for one week; ran away from hospital; sent to House of Detention
14.	G. H.	4	M	When 2 years old he was dropped on pavement, injuring his head; outside physician diagnosed concussion and stated did not believe he would be mentally normal if he recovered	Total change in behavior; delights in torturing animals, scratching and pinching people; has unusual energy; in clinic uncontrollable, cried, knocked furniture over, had terrific outburst of temper and had to be taken home without examination; home visit Oct. 28, 1923, still unmanageable and noisy	....	Traumatic constitution	Unimproved
15.	S. K.	6	M	Struck on head when 5 years old; convulsions followed	Violent temper; irritable; very noisy; breaks things; stupid	....	Traumatic constitution	Unimproved
16.	M. K.	10	F	May 5, 1916, struck by automobile; brought to hospital in stuporous condition; blood pressure, systolic 90, diastolic 60; slow pulse; diagnosis, concussion of the brain; discharged from hospital May 17, 1916	Unable to progress in school; three years in one grade, then demoted to special class	....	Posttraumatic defect; mental deficiency	Has been removed from city school and placed in country with a good caretaker; excellent physical condition; teacher states child is dull but eager to learn; unable to concentrate for more than a few minutes at a time

TABLE 2.—*Summary of Cases—Continued*

Name	Age	Sex	Date and Type of Injury	Neuropsychiatric Sequelae	Roentgen-Ray Findings	Diagnosis	Present Condition
17. G. M.	13	F	September, 1919, hit by automobile, thrown down; head injured in January, 1921; struck by automobile again, patient fell on back, automobile ran over her; unconscious	General change; nervous; change in character; at times very emotional; period of typical chorea; not getting along as well in school; occasional headaches, some dizziness	....	Traumatic condition	Improving
18. G. M.	12	M	When 8 years old struck on head by brick; necessary to suture scalp	When 2 years old child was thought peculiar, but after accident seemed to be a complete change of disposition: from being stupid and retiring became overactive, untruthful, ran away, had violent outbursts of temper and would steal; quarrelsome; intelligence quotient 65	....	Mental deficiency, posttraumatic in origin	Placed by Society in country home; ran away several times; finally arrested for stealing and sent to juvenile court
19. J. R.	12	M	Struck by automobile as he was crossing street, April 14, 1917; admitted to hospital in dazed condition; discharged from hospital later in good condition	Unable to concentrate; unable to progress; inattentive in school; slow in movements; frequently wakens during night crying	....	Traumatic constitution	Two years retardation; anemic; fights, selfish, stubborn, irritable
20. J. N.	11	M	Fell, at age of 2 years, out of a window, striking head; eyes crossed	Conduct difficulties in school; violent outbursts of temper; nervous habits	No demonstrable bone lesion..	Posttraumatic condition	Not known
21. J. R.	16	M	Hit by base-ball but when 5 years old; unconscious; not irrational when taken to hospital; 1 hour later generalized convulsions	Marked depression of skull; fracture extended well back toward center of head and was circular in outline, about 1½ to 2 inches in diameter; slight abrasion over the mass; edema under skin; decompression operation performed on date of admission	....	Fracture of skull; posttraumatic defect	Unimproved; at times sleeps 35 hours at a stretch; recently stole $83 from employer; lacks moral sense; right pupil sluggish and pulse averaged from 50 to 60; eye examination negative
22. N. S.	14	M	At age of 8, fell backward striking head on cement pavement; unconscious for few minutes and seemed to recover from fall	One month later began having attacks during which he raised left arm, frequently made queer sounds and groaned; spells at intervals of four weeks for next two years, attacks always occurring on left side; behavior abnormalities; quarrelsome; temper tantrums; arrested for breaking windows; later, generalized seizures	Negative..............	Traumatic defect with Jacksonian attacks	Referred to surgeon who advised against operation; no change
23. H. S.	?	F	June 16, 1922, struck on top of head by falling cellar door; brought to hospital unconscious	Mischievous; resents discipline; no fear of danger; before accident in second grade; remained in second grade since	Negative; traumatic rupture of both drums of ears; neurologist reports right facial paralysis; discharged July 10, 1922	Right facial paralysis following trauma; traumatic constitution	After 16 treatments of electricity, was discharged from clinic as well; facial paralysis cleared up entirely; continued having discharge from both ears; improving

No.	Initials	Age	Sex	Accident	Finding	Symptoms	Diagnosis	Outcome
24.	A. S.	13	M	When 2½ years old had severe fall from chair; unconscious following fall; did not talk for two months afterward		Afraid of everything; gets into rages; steals money; violent temper; unable to progress beyond fourth grade; restless, dishonest, forges checks; wanders away from home; sent by police on many occasions to House of Detention; talks foolishly; throws things when "mad"; flippant; frequently refuses to answer questions; intelligence quotient 71	Traumatic constitution	After several arrests child was placed in Junior Carter Republic and under supervision and discipline is doing well
25.	G. S.	13	M	Accident when 10 years old; struck by automobile on head	Negative	Stealing from parents; untruthful; disobedient; poor judgment; unable to progress in fourth grade of school; conduct in school very bad; annoys other children by constant talking and teasing	Posttraumatic behavior disorder	Sent to truant school; unimproved
26.	F. W.	9	M	Sept. 27, 1923, fell out of third story window, striking head on pavement; brought to hospital unconscious		Paralysis both sides of face; mentally very confused; disoriented; examination incomplete on account of overactivity and resistiveness of patient; neurologic: right-sided Babinski, abdominals absent on right	Traumatic defect	Unknown; unable to get a report of his condition since family moved
27.	S. W.	9	M	When 5 years old, struck by bicycle; thrown to pavement; striking forehead; dazed	Negative	Talks foolishly; cries easily; dishonest; cannot be disciplined; "crazy about fire"; headaches; unable to be sent to school until 8 years old and is still in first grade	Mental deficiency, posttraumatic in origin	Strabismus; eye report shows mild astigmatism; after 1 year in country seems "not so nervous"; still in first grade; emotional lability; country life has apparently done much for him
28.	B. W.	11	F	When 1 year old, fell out of crib and struck head on floor; unconscious; had convulsions at intervals of from 3 to 4 months		Never been normal since fall; has spells when she talks incoherently; silly manner; slow to learn	Imbecility; epilepsy, posttraumatic in origin	Sent to institution for feebleminded, where she is making slow progress
29.	H. V.	14	M	Fell off the bars in school-yard, hurting head in 1917		Marked change in character; in school is defiant, disobedient, annoys the other children, extremely quarrelsome, attempts to strike whenever reproved; intelligence quotient 66; probably due to affective reaction	Traumatic constitution	Improving; continues to show marked emotional instability characterized by irritability and quarrelsomeness
30.	L. K.	13	F	At age of 5 years, struck on back of head with a large iron pistol	No demonstrable evidence of skull fracture	Lost her speech and the use of her limbs for 6 months; has been going backward in school work; cruel to younger brothers and sisters; lies on roof in rain; hard to control; obstinate; violent fits of temper; eats out of garbage pails; one cold night family found her stripped with bottle of water tied tightly around neck; another time when family had retired, dressed and went out in the street, returning at midnight; wets bed every night; threatened to kill people	Posttraumatic defect, periods of epileptic equivalent	Continues having temper outbreaks; extremely emotional, unstable; at times paranoid attitude; at other times complains of depression; states she will take her life; hospital admission advised

chronologic age progressed to seven with but two repetitions. It also seemed that there was a qualitative difference in performance as those with traumatic cases frequently transposed the digits. It was felt by the examiner (Marie W. Peters, Clinical Psychologist) that the number of repetitions required might be a significant index of the fatigability. The results are given in the accompanying table.

COMMENT

The cases may be considered briefly from the following points of view: first, similarity to postencephalitic behavior disorders and the contrast to adult traumatic sequelae; second, relationship to problems of delinquency; third, prognosis and treatment.

The behavior disorders described bear a marked similarity to those common after acute epidemic encephalitis in children. One of us,[5] in writing of the sequelae of encephalitis, stressed particularly the total change in general character and disposition, with hyperkinesis and affective disorders such as we have here noted. Since both conditions are really organic reactions, we might expect this similarity. As in encephalitis, we also find in the post-traumatic group that the mental reaction may often represent an accentuation of the previous personality make-up. This point has been adequately emphasized by Adolf Meyer.[6]

The findings should lead to caution in referring these conditions to hypothetical molecular changes in the brain. We would prefer to think of the problem in terms of the total reactions of the individual child. A study of the child, the situation and environment, and the reaction to this situation and environment before and after the injury is therefore imperative.

In comparison with sequelae in adults one notes the absence of traumatic neuroses. The desire for compensation and the effect of litigation procedures played no rôle in this group of children. Likewise, intolerance to alcohol need not be considered. The affective lability, vasomotor changes and epileptoid states are similar.

These disorders have a definite connection with delinquency, and indicate the need for detailed neuropsychiatric study of all children of this type. As the majority of our patients were referred from the schools, the importance of giving educational authorities data concerning the traumatic group is obvious. The effect of explosive behavior disorders in the school room naturally adds to the teacher's difficulties as do the fatigability and attention defects; all of them indicate the need of different educational requirements.

5. Ebaugh, Franklin G.: Neuropsychiatric Sequelae of Acute Epidemic Encephalitis in Children, Am. J. Dis. Child. **25**:89-97 (Feb.) 1923.

6. Meyer, Adolf: The Anatomical Facts and Clinical Varieties of Traumatic Insanity, Proc. Am. Medico-Psychological Assn., Washington, D. C., May, 1903.

There are few statements in the literature concerning the prognosis. English,[7] however, says that "the seriousness of severe head injuries in children is determined by their occurrence at a time when the intellectual faculties are being developed." He considers cranial injuries in youth and old age as equally poor in outlook. In our group we can only state conservatively that six are improving, whereas the outlook in the remaining twenty-four is discouraging.

Treatment consists in careful surgical management during the acute manifestations of cerebral trauma. Wilensky,[8] Sharpe[9] and others write of the advisability of this step. Rest from physical and mental exertion, removal to a quiet, nonirritating environment, intensive study in mental hospitals, more individual consideration in school, special classes and vocational guidance, are all matters of importance in treatment. Appropriate surgical interference in patients showing jacksonian seizures is clearly indicated in some instances. During periods of mental excitement, we find that drugs are of little avail, but hydrotherapy is often helpful.

7. English, T. C.: Lancet **1**:845 (Feb. 20) 1904.

8. Wilensky, A. O.: (Footnote 1).

9. Sharpe: The Diagnosis and Treatment of Brain Injuries, Philadelphia, J. B. Lippincott Company, 1920.

Abstracts from Current Literature

SOME SIMPLE METHODS FOR THE STAINING OF NEUROGLIA CELLS. S. RAMON CAJAL, Schweiz. Arch. f. Neurol. u. Psychiat., Festchr. f. Constantin von Monakow **13**:187-193, 1923.

Most of the stains for neuroglia cells present a great deal of difficulty, and Cajal wishes to draw attention to some simple procedures of impregnation which have already been published but which posses distinct advantages.

Gold Chlorid Method.—1. Fixation of fresh pieces for from two to twenty days in a solution of:

Neutral formaldehyd	15
Water	85
Ammonium bromid	2

2. Frozen sections, from 25 to 30 microns thick.

3. After rapid washing in distilled water, the sections are put into a solution of:

Distilled water	0.35 c.c.
Solution of gold chlorid, brown (Merck) 1 per cent...	6 c.c.
Sublimate crystals	from 0.5 to 0.8 gm.

The sublimate is dissolved in the gold solution on warming. It is well to filter, and it may be used cold. No more than four or seven sections from 3 to 4 cm. surface area should be used to 35 c.c. Watch crystals may be used to advantage. After the sections have remained in this solution for from four to six hours at a temperature of 18 to 20 C., they will be found to have taken on a purple hue.

4. By means of glass lifters they are passed through large amounts of distilled water and then placed for ten minutes in a solution of:

Sodium hyposulphite 5 per cent	0.40 c.c.
Alcohol	10 c.c.

5. They are next washed in a 30 to 40 per cent. solution of alcohol, mounted, the excess liquid removed with blotting paper, dehydrated with absolute alcohol, oil of origanum, or carbol-xylol and imbedded in xylol balsam.

For the success of the method, the reagents should be pure and the solutions fresh. Tissues from all vertebrates are well stained. While the gold solution, protected from light, keeps well, it deteriorates rapidly after mixture with the sublimate. In general, the increase of the proportion of the sublimate up to double or triple of the amount specified accelerates the reaction but detracts from the beauty in increasing the size of the granules. Higher temperatures diminish the contrast of the coloration. The fixation in formaldehyd gives best results when carried on for a period of three days, but may be prolonged to twenty days. The protoplasmic substance quickly loses its staining powers; however, the fibers preserve this for many years. One of the defects of the method is the failure to give more than a pale coloration to cells in the molecular layer and in the layer of small pyramidal cells. This is probably due to the action of the formaldehyd, and may be obviated partially if the pia mater is left in position. Fresh cases of encephalitis yield excellent results.

Ammoniated Silver Oxid Method with Formaldehyd-Bromid Fixative.—This method depends on the affinity for silver communicated to the glia cells by ammonium bromid. It has the advantages of simplicity, strong coloration and reliability. The method is not to be surpassed for the impregnation of astrocytes; for the presentation of protoplasmic glia cells, it is as good as the preceding method.

1. Fresh pieces are fixed for from three to fifteen days in solution of:

Distilled water	85 c.c.
Formaldehyd	15 c.c.
Ammonium bromid	2 gm.

2. Frozen sections of from 15 to 25 microns are received in the same fixative. They are transferred to the following mordant for forty-six hours at a temperature of from 30 to 38 C., or for from eight to ten hours at room temperature:

Formaldehyd	6 c.c.
Ammonium bromid	3 gm.
Water	50 c.c.

3. Wash twice in distilled water.

4. Transfer to a solution of ammoniated silver oxid prepared according to the methods of Bielschowsky, Achucarro, Rio-Hortega, or the following:

Distilled water	10 to 15 c.c.
Ammoniated silver oxid	5 c.c.
Pyridin	4 to 5 drops

5. The bath containing the sections is heated in a crystal until the sections become the color of tobacco. The temperature must not be excessive, and to avoid this the flame should not touch the crystal and should be removed from time to time so that the solution may cool.

6. Wash rapidly in distilled water, leaving them no longer than from three to five seconds.

7. Reduction in formaldehyd of 5 per cent., from two to three minutes.

8. The impregnation is improved, and the sections are rendered more transparent by placing them in a solution of yellow gold chlorid, 0.2 per cent. This treatment is absolutely necessary for the thicker sections. The excessive gold chlorid solution may be removed by a solution of sodium hyposulphite, 5 per cent. for five minutes.

9. Wash, dehydrate as above, and mount in balsam or damar.

The stain will not succeed if the material is old, if it has been fixed more than ten days, if the solution of silver oxid is too dilute, or if the washing under 4 is continued too long. Not only is the glia substance stained, but also the vascular and extraparenchymatous connective tissue, and the Nissl bodies are tinted. Connective tissue of neoplasms stains constantly.

Uranium Formaldehyd.—This method is particularly applicable to the demonstration of Golgi's reticular apparatus. The method is superior to the uranium acetate method of Rio-Hortega or the cobalt nitrate method of Da Fano. Failure to obtain results is due to excessive thickness of the blocks or failure to follow the technic. The value of the method lies in the absolute constancy with which gliosomes and lipoid inclusions are demonstrated.

1. Fresh tissue of not more than 3 to 4 mm. thickness is fixed in from twenty-four to not more than forty-eight hours, in the following solution:

Uranium nitrate	1 gm.
Neutral formaldehyd	15 gm.
Water	85 gm.

2. Wash rapidly, submerge the pieces in 1.5 per cent. silver nitrate solution for two or more days at room temperature.

3. Rinse for several seconds in distilled water and reduce in the following bath:

Hydroquinon	1 gm.
Formaldehyd	10 c.c.
Sodium sulphite sufficient to render the liquid a yellowish color.	

4. After twenty-four hours, wash, dehydrate and imbed in celloidin.

If the pieces are placed in the fixative for less than from ten to twelve hours, the reticular apparatus of Golgi appears in lieu of the neuroglia tissue. The method gives excellent results in almost all vertebrates. The superficial sections are utilized. Sometimes only the gliosomes and inclusions are stained, but the neuroglia protoplasm remains unstained when sections are taken from the deeper parts of the block. The nuclei may be counterstained by some basic blue or violet anilin dye.

The neuroglia cells of the gray substance differ from those of the white substance in that the former have a spongier structure, intraprotoplasmic gliosomes, a dentate appearance of the long processes and a material in the spongioplasm which disappears rapidly when the tissue is fixed in formaldehyd. These characteristics are not noted in the neuroglia cells of the white matter. The processes of astrocytes are seen to be numerous. Every glia cell is seen to possess one or more processes by which it attaches itself to the perivascular structures.

WOLTMAN, Rochester, Minn.

A CYTOARCHITECTURAL ATLAS OF THE BRAIN STEM OF MACACUS RHESUS. AUBREY T. MUSSEN, J. f. Psychol. u. Neurol. **29**:30, 1923.

Continued disappointment and confusing results after experimental procedures convinced the writer that careful, scientific data could be obtained only after the ground had been prepared by exact and exhaustive study of the forms on which the experimentation was to be carried forward.

The difficulties of correctly localizing the structures to be approached experimentally soon became evident, and the author applied himself to the preparation of a series of brain sections made in three different planes, by means of which he would be able to locate accurately in space the structure to be approached.

The present atlas was completed in 1914, but owing to difficulties in publication incident to the war, its appearance has been delayed until the present time. The illustrations are original.

The method for the localization of the structure to be experimentally attacked is based on the establishment of planes in the sagittal, horizontal and frontal directions. By careful measurement, the structures can be located in relationship to these three planes, and then by proper adjustment of the stereotaxic apparatus, the stimulating or destructive point can be introduced with exactness.

The three basal sections, that is, sagittal, frontal and horizontal, are established as follows: A basal, horizontal line is drawn from the lower margin

of the orbit to the middle of the external auditory meatus. Ten millimeters above and parallel to this basal horizontal line is then drawn a second line called the zero horizontal plane. A zero frontal plane is then established by passing a plane at right angles to the already established basal line through the external auditory foramina. The zero sagittal plane passes through the fissure longitudinalis perpendicularly.

The method of marking these lines in the brain in situ within the skull is then described. This is done by passing a needle dipped in ink and mucilage through the skull into the brain, leaving the ink marks in the brain substance itself. The method of fixation of the brain is described.

The brain should be measured before and after fixation in order to make proper allowances for shrinkage. By means of these three established planes, the exact location of any structure within the brain stem can then be established by careful measurement. If the sections are cut in the frontal plane, the zero horizontal plane already indicated is marked in a similar manner to that outlined above, the median line indicating the zero sagittal plane and the number of the section indicating its relationship to the zero frontal plane. If the sections are cut in the sagittal plane, the zero and basal horizontal planes are marked in the manner indicated above.

The location of any structure within the brain stem to be approached experimentally can be carried out by locating the anatomic limits of the structure to be attacked. Its distance from the zero plane and by means of calculations, the exact position and the procedure necessary to the proper location of the stimulating or destroying point of the stereotaxic apparatus can then be carried out.

If an investigator wishes to determine the actual position of any structure within the brain stem, it is necessary for him to determine the sections in which the structure first begins to appear and that in which it finally terminates.

The calculation by which the actual size of the structure of the stem can be obtained depends on the limits of the structure as obtained by the sections and on the enlargement used, the reduction by means of the proper denominator giving the actual size of the anatomic structure.

After the location of the structures to be experimentally approached has been established by means of these zero planes and after the correct measurements of the head have been obtained, the stereotaxic machine can be adjusted in such a way that the stimulating or destroying point can be made to approach accurately any desired part or structure of the brain stem.

Accompanying the article describing the methods of locating any desired structure within the brain stem is an atlas giving a set of nineteen frontal sections from the region of the corpora quadrigemina and the inferior olive, caudally, to the anterior commissure cephalically. There is in addition a set of fourteen sagittal sections including that part of the brain which lies between the superior olivary nucleus and the nucleus caudatus and from the medial line to the lateral limits of the thalamus. Each plate is marked with lines giving the position of the sections cut in the other plane. The illustrations are profusely indexed with numbers marked on them which correspond to the various anatomic structures represented in the sections. The latter is accompanied by a key which indicates the name of the structure marked by a number on the plate and accompanied by a fairly elaborate description. Similar structures appearing in a number of successive plates are always identified by the same numeral.

At the close of the article there is an alphabetical and numerical index of all the structures figured in all of the plates.

A second part of the article is devoted to a careful, cyto-architectural analysis of the thalamus. Although this study is cyto-architectural, the auhor has adoped the myelo-architectural classification of the thalamus adopted by Vogt. This is rendered possible by the close similarity of results obtained by these two methods of study.

The author mentions the interesting fact that the various cell groupings in the thalamus are determined chiefly by the fiber distribution, which fact the reviewer has had abundant opportunity to establish in his work on the thalamic nuclei in the primate brain stem (at present in course of preparation).

It has, however, been increasingly evident that both of these methods are somewhat open to criticism in that in both the cellular pictures and in the myelinated sections the transition is so slow and gradual between the pulvinar and the lateral nucleus of the thalamus particularly, that the exact point of transition between these two structures is almost indistinguishable. The delimitation of the pulvinar from the thalamus proper is demonstrated in considerable detail in these structures by means of the plates, but the reviewer is at a loss to be able to establish definitely the lines of transition between these two structures. The cellular groupings making up the nuclei of the thalamus are indicated extensively. It is, however, extremely difficult to follow the limitation and transition pointed out as taking place in the conformation of the nuclei.

The plates are faint, and the magnification so low that details can be accepted only as given by the author. Nuclei are indicated as succeeding other nuclei without any clear and definite, demonstrable differentiation. Bundles of fibers are indicated by number, but no actual demonstration of fibers is made. The method of examination is entirely by cellular stains. The plates are most painstakingly studied and lettered, and the atlas represents an enormous effort and should be of great value to any one studying this region. Its value would have been greatly enhanced if the cyto-architectural plates could have been paralleled by fiber stained preparations.

RILEY, New York.

VESTIBULAR NYSTAGMUS. MECHANISM OF ITS PRODUCTION IN NORMAL AND PATHOLOGICAL CONDITIONS. BETCHOV, Rev. neurol. **30**:209 (March) 1923.

The greatest difficulty in the study of nystagmus is the attempt to compare or reconcile the existence of a normal nystagmus with that found clinically in pathologic states where there is an exact or precise anatomic lesion. In other words, nystagmus produced by whirling movements, hot and cold douchings of the ear, the galvanic current, etc., apparently has nothing in common with nystagmus found, for example, in multiple sclerosis. The one appears in a healthy person whose semicircular canals are stimulated in some particular way; the other appears in disorders which have other pathologic signs, such as ankle clonus, intention tremor, dysmetria and the like.

In 1918, the author offered an hypothesis to the effect that nystagmus in pathologic conditions is simply one of several manifestations of increased motor activity. This view is inadequate, however, in the light of later investigations, which showed that nystagmus was fundamentally an ocular reflex rather than an hypermotricity.

Bard, as early as 1904, had spoken of "the vestibular chiasm, or the decussation, at the level of the encephalic trunk, of the afferent pathways in connection with the perception of gyration."

In his earlier studies, the author, for lack of an established anatomic basis, had fallen back on the idea of a "reflex of conjugate deviation of the eyeballs." This deviation was regarded as due to decussating afferent pathways in a manner analogous to the decussation of the fibers of the optic chiasm. The double labyrinthine apparatus perceives nothing but movements (just as the eye takes in color and form). On the other hand, the vestibular chiasm concerns itself with perceptions where special characteristics are concerned, similar to the optic chiasm.

Bard's leading idea is that here is a sort of hemilabyrinthine nerve or hemivestibular fillet analogous to the bundle of optic fibers of the same name in either half of the cerebral peduncle and of the protuberance. In other
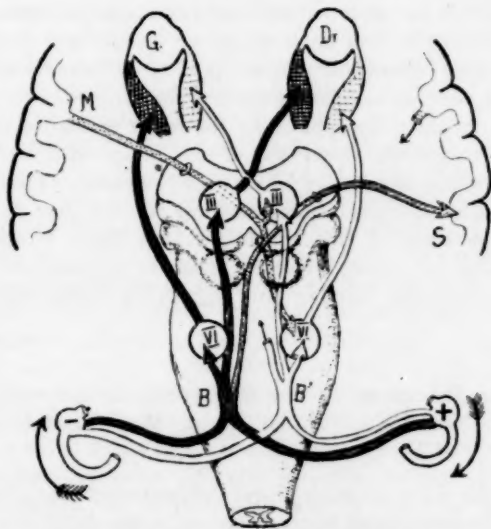
Illustration of author's hypothesis.

words, sensations of rotation toward the right and of falling toward the left pass along fibers in the left half of the cerebral peduncle, and vice versa.

Nystagmus appears in the light of this synthetic conception as constituted: First, by a reflex of conjugate deviation of the eyes opposed to the sense of perceived movement. The reflex of conjugate ocular deviation constitutes the slow phase of nystagmus and is its most essential phenomenon; it is the most persistent as well as the most primitive appearing in narcosis and in the very young. In the new-born, this slow conjugate movement accompanies rotation of the head. In the adult, it is still accompanied by a general orientation of the body as regards sense of motion or postion. Second, the rapid phase of nystagmus helps us to determine the direction of nystagmus proper. This rapid phase or "clonic" movement is difficult to explain. It is probably a compensatory movement associated with movements which the head undergoes incessantly in the course of all locomotion, active or passive. It is comparable to the end result in the physiology of sight, namely, the efficacy of getting as perfect a visual image on the retina as is possible.

The interpretation of this rapid movement in nystagmus is the real aim of the paper. The author offers a theory which has certain advantages of simplicity and clarity as compared with those offered by earlier writers. He first refers briefly to Coppez's hypothesis of a subcortical center of coordination, and to Bard's idea of a spastic conjugate movement, intermittent, a dysmetria, a reaction of certain centers of motor inhibition. From these he passes to his own hypothesis illustrated by a conventional drawing.

The arrows mark the direction of rotary movement; the concomitant impressions, bilabyrinthine, of rotation to the right, due to some kind of pressure (hot or cold water, galvanism) converge in the left hemivestibular fillet and produce levogyric ocular deviation. Extension or flexion of the head in various degrees produce various modifications. Rotation of the head or trunk to the right, actual, or induced by irrigation, affects necessarily, in a contrary manner, the nerve terminations which have their pathways in the right fillet, and throw the right oculogyric system into hypertonia without which it is impossible to arouse a cerebral or cerebellar influence. The right oculogyric nuclei, deprived of their tonus of repose, are then stimulated to states which produce dysmetria under any and all circumstances. If these views be accepted, then nystagmus assumes a significance most simple, namely: It is a conjugate deviation brought on by the reflex excitation of an oculogyric system associated with the hypotonia of an antagonistic system, the latter resulting in a dysmetria which may be cortical (voluntary movement as opposed to the slow phase), subcortical (habitual tonus of ocular fixation) or even peripheral. In the light of this hypothesis, the three forms of nystagmus (horizontal, vertical and rotary) find an adequate explanation. Further study is necessary to determine whether this hypothesis holds where pathological cases obtain.

JONES, Detroit.

A NEW OPERATIVE PROCEDURE IN THE TREATMENT OF SPASTIC PARALYSIS AND ITS EXPERIMENTAL BASIS. N. D. ROYLE, M. J. Australia **1**: (Jan. 26) 1924.

Following on the work of Bocke, who demonstrated that voluntary muscles are supplied by nonmedullated fibers from the sympathetic system in addition to the medullated fibers from the anterior horn cells, Royle conducted a series of experiments to determine the effect of cutting the sympathetic nerve trunks on the spasticity in the limbs produced by decerebration section or hemisection of the spinal cord. Experiments were conducted to show the function of the sympathetic fibers supplying the voluntary muscles and whether that function had any relationship to the abnormal muscular condition encountered in spastic paralysis. The removal of the abdominal sympathetic nerve trunk produced a similar effect in every experiment regardless of whether the peripheral spasticity was produced by decerebration complete or hemisection of the spinal cord. Simple removal of the trunk did not interfere with the animal's ability to control the lower limb on that side, but the animal when placed on its back was not able to maintain the limb in an extended position, and the amplitude of the tendon jerks was diminished. In every instance, the observations indicated that there was a definite change in the lower limb when compared to the normal limb of the opposite side, and that change may be described as a depression in reflex activity and a disturbance of the mechanism for maintaining posture of the limb.

The hypertonicity and the flexion following transverse section of the cord were profoundly altered in the lower limb after section of the abdominal sympathetic trunk on that side. In contrast to the spasticity existing on the opposite side, the limb fell into extension and abduction under the influence of mechanical factors, while the knee and ankle reflexes were less active. Under the conditions of decerebrate rigidity, the division of one abdominal sympathetic trunk prevented the onset of the rigidity in the lower limb on that side, but the limb participated in the periodical extending movements only to fall into flexion immediately while all other limbs remained in extension. During the intervals when rigidity was less evident, the right and left lower limbs still exhibited a striking contrast. After section of the left abdominal sympathetic, the right limb usually maintained a semiflexed attitude through the period of decerebration, and the knee reflex was readily maintained and exaggerated in character. The left lower limb, on the other hand, fell into a more flexed attitude, and while the knee reflex was obtainable, it was decidedly less in amplitude. If, however, the left lower limb were put into a similar position to that assumed by the right lower limb, the knee reflex exhibited about the same amplitude. These conditions were best developed in an animal in which the abdominal sympathetic trunk was removed at a considerable time before decerebration was performed.

The operation of sympathectomy was performed twice on human subjects. In the first case following a gunshot wound of the head with destruction of the left motor cortex, a spastic hemiplegia affected both limbs on the right side. In the right leg, there was an exaggerated knee reflex, a patellar clonus, an ankle clonus and an increase in the ankle reflex. The patient could walk, but had the greatest difficulty in using the right lower limb, which he moved as one rigid piece. When he placed weight on the limb, there appeared a coarse uncontrollable tremor. He could not stand on the right lower limb, although he could stand comfortably on the left. It was only possible for him to move forward; lateral or backward walking could not be carried out. There was present also a slight degree of talipes equinus on the right side.

The right abdominal sympathetic was removed by a posterior extraperitoneal approach. The only operative difficulty was the depth of the incision. The sympathetic trunk was not difficult to recognize. It was removed from the second to the fifth lumbar nerves. The improvement in the patient's condition was remarkable. Twenty-four days after operation, he could walk up and down a flight of steps, using each leg alternately, stand on his right leg alone and walk backward or laterally. The knee and ankle clonus disappeared from the right side. There was little difference in the degree of amplitude of the knee and ankle reflexes on the two sides. The vasomotor dilatation which had been noted on the right side had practically disappeared.

In the second case, a spastic hemiplegia had existed for fourteen years. The upper limb was useless, as there was only the slightest degree of voluntary control in the flexor profundus of the fingers. The hand was rigidly dorsiflexed at the wrist. The rigidity was so severe that it was impossible to flex or extend a finger passively, and the extensors of the wrist resisted forceful passive flexion. The wrist reflex was exaggerated and followed by a clonus.

At operation, the cervical sympathetic was avulsed from the fifth cervical to the first thoracic nerves. Immediately after operation, it was evident that the former rigidity had been very much diminished. It was possible to flex the wrist passively without any resistance from the extensor muscles. The

fingers could be flexed and extended at the interphalangeal joints, but could not be extended at the metacarpophalangeal joints on account of structural changes. The forearm could be supinated without resistance through an angle of forty-five degrees. The patient could voluntarily dorsiflex the wrist joint and supinate the forearm. The reflex activity was much diminished, although the wrist reflex was still easily elicited. There was a transitory vasomotor disturbance on the operated side.

In both these instances, there has been a remarkable diminution of rigidity with such an increase in the ability to control movements that the procedure should be effective in practically all cases of spastic paraplegia and hemiplegia. The best results will be obtained in those patients whose movements are present, but are rendered defective on account of rigidity, although the diminution of ridigity will probably enable movements to be performed that were quite impossible before. In spastic paraplegia of infancy, the difficulties of balance would be very much lessened after section of the sympathetic nerves. In addition, the diminution of rigidity would enable the patient to make effective whatever cerebral control happened to be present and should assist materially the process of education. In those instances in which hemiplegia comes on as the result of trauma or disease and the paralysis is not progressive in nature, similar results should be obtained, in all probability much more readily, as in these cases. The process will be one of reeducation only.

GRANT, Philadelphia.

THE SUBENDOCARDIAL GANGLION AND NERVOUS CELLS; THEIR RÔLE IN CARDIAC AUTOMATISM. M. EIGER, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow 13:223-232, 1923.

While numberless efforts have been made to solve the problem of cardiac automatism, it is not entirely settled. As yet neither the neurogenists nor the myogenists have been able to furnish direct experimental evidence with which to prove their contentions.

The experiment of Carslon, 1904 to 1908, on the heart of *Limulus polyphemus,* the horseshoe crab, are considered by Tigerstedt to be a direct proof of the theory of neurogenic innervation for the lower invertebrates. The heart of *Limulus* is from 12 to 20 cm. long and 2.5 cm. wide. Carlson found that there were in this heart three trunks, one median and two lateral. The median trunk was made up of a mass of nerve cells united with each other by nerve fibers, thus constituting an elongated ganglion; the lateral trunks were without nerve cells. Extirpation of the median ganglion will arrest the heart in diastole. For this reason, Tigerstedt does not hesitate to attribute a prominent rôle to this ganglion, and believes that this fact furnishes direct proof of the theory of nervous innervation. Carlson has, furthermore, shown that the embryonic *Limulus* heart will contract when there are no nerve cells present. At this period of its evolution, the structure of the heart is syncytial and without fibrils or striations. After the appearance of the nerve cells, the contractions lose their embryonic character, and the automatism of the heart is exclusively dependent on the nerve cells.

This is in perfect analogy with the trunkal muscles of *Selaciens* (sharks) studied by Wintrebert (1917 to 1921). This author demonstrated that the movements of embryonic *Selaciens is* not dependent on nervous innervation, but is idiomuscular, or aneural. After an intermediate period the movements are placed under the domination of the nervous system. Winterbret has shown

that one can cause the idiomuscular type of activity to reappear after ablation of the cord. Cardiac muscle therefore presents no exceptions, and a classical example of this is found in the *Limulus* heart.

Minkowski, who studied the movements and muscular reactions of the human fetus of 2 to 5 months and their relation to the fetal nervous system, demonstrated the coexistence of motor phenomena of different orders. He showed, first, motor phenomena of a nervous type, dominated by the central nervous system, which were characterized by extreme variability, the faculty of generalization and a tendency to assume a particular biologic character; and second, direct aneural excitability of the muscle which persists after those of nervous origin have disappeared, following ablation of the cord, which present a more massive, more localized and more stereotyped character.

In 1911, Eiger observed in thionin-stained serial sections of the hearts of white rats, of rabbits and of man, first, that the largest number of ganglion cells are found posterior to the left auricle, secondly, that others exist between the openings of the vena cavas (ganglions of Remak), thirdly, others in the interauricular septum (ganglion of Ludwig), and fourthly, in the transverse coronary fold. All of these ganglion cells are found in the connective tissue of the subpericardial, or more exactly subepicardial, region. They are regulatory and related to the parasympathetic system, as Englemann and Langley have shown.

In 1918, Eiger attempted to correlate these findings with the nervous theory of cardiac automatism. If the opinion of Magnus is correct, that the plexus of Auerbach of the intestine is a motor plexus, and that its ablation causes the arrest of peristalsis, it is reasonable to assume that the automatism of cardiac movements is also dependent on nervous structures. For this purpose the hearts of the snail (*Helix pomatia*) and of the frog (*Rana esculenta*) were selected. Some writers describe ganglion cells in certain molluscs, whereas others deny their existence. This diversity of opinion is probably dependent on differences in technic; it is not only necessary to make serial sections, but the direction of the sections in relation to the axis of the heart is important, since ganglion cells may easily be missed in transverse sections. In the study of sections of the frog, the ganglions of Remak, of Ludwig, and of Bidder, described long ago, are easily recognized. The epicardial ganglion cells were easily found. In addition to these, another group of nerve cells which lie subendocardially within the interior of the venus sinus, were seen, which the author has not found described before. In the region where the right anterior vena cava comes in contact with the venus sinus, a high endocardial plica is found. This fold, placed on the right side of the opening of the sinus, directs the current of the blood from the right superior vena cava toward the top, and consequently opposes the blood flow toward the sinus. It is in this fold that Eiger found a group of ganglion cells placed in the subendocardial connective tissue. To the left of the venus sinus and under the osteum, isolated nerve cells, also subendocardial, may be seen.

After having sectioned the pericardium of a frog, and with the aid of a loop, the anterior wall of the posterior vena cava and of the venus sinus was incised. The osteum appeared like an orifice against which the blood was thrown with each contraction of the auricles. Every time this occurred, the plica endocardialis resembled a bleached triangle. When this fold was touched with a thermocautery and these nerve cells as well as those to the left of the osteum were destroyed, the heart was arrested in diastole. It was necessary to touch this fold only lightly. When only the cells of the fold were destroyed,

one noted, after complete arrest of the heart, a few small contractions of certain muscular fibers to the left of the sinus, whereas those on the right remained immobile.   After from twenty-five to thirty minutes, the heart recommenced beating.   The heart beat was again arrested when the nerve cells situated to the left of the sinus were destroyed.   Therefore, in order to obtain complete arrest of the heart, it was not only necessary to destroy the principal group of cells contained in the fold, but also to destroy the cells which are isolated in the subendocardial structure to the left of the sinus.   The arrest of a heart after destruction of the subendocardial nerve cells also results in an atropinized heart, the inhibitory action of the vagus nerve not interefering.

These experiments seem to constitute a direct proof of the important rôle which the subendocardial ganglion cells play in the cardiac automatism of a frog.   They furthermore demonstrate the motor function of these cells.   The refractory period is given as the cause of rhythmic movement.

WOLTMAN, Rochester, Minn.

STUDIES ON THE FINER ARCHITECTURE OF REGIONAL CORTICAL AREAS IN RODENTS. 1. THE SUBOCCIPITAL CORTEX (BROADMANN'S RETROSPLENIAL CORTEX). S. R. y. CAJAL, J. f. Psychol. u. Neurol. **30,** Nos. 1 and 2 (July) 1923.

In this scholarly article, Cajal amplifies his earlier studies, made in 1893, on the same subject.   He emphasizes his belief that identity in architecture corresponds to identity in dynamic function, and that the finer structural criteria should prevail over topographical desiderata.   Yet in rodents there exist several cortical areas possessing genuine Gennaris stripes and granular layers as they are wont to occur in the visual lobe, although these areas are not concerned with vision.   Hence he feels that the anatomico-physiologic specificity of a given brain area should be the result of the concord of three requirements: (1) The finer structure of the strata in the gray matter; i. e., the intimate neural morphology, if possible with the origin, course and end of the neuron; (2) physiologic and experimental pathologic data; (3) ontogenetic studies respecting the progressive medullation of fibers.

Cajal recommends the use of the brains of young (fifteen to thirty day old) animals, using the silver methods (Golgi-Cox) and neurofibrillar stains rather than adult brains.

His recent critical analysis of the structure of the rabbit's suboccipital cortex (Brodmann's retrosplenial area) follows:

1. The plexiform or molecular layer.   This is comprised of (a) an inner zone of spindle-shaped pluripolar type cells lying horizontally, close to the perpendicularly disposed spindle cells of the next layer.   Their polar branches are long and send off fine horizontal branches which strike off at right angles to the parent stem and resemble axis cylinders.   Many ascending fibers end in this layer and form a thick network about these cells.   Some are terminal ramifications of fibers arising from deeper layers and even from the white substance.   Again one finds stout fibers running horizontally possessing thick medullary sheaths.   (b) An outer zone containing many branched fibers enmeshing cells of Golgi, Types 1 and 2.

2. Zone of star cells.   These are star-shaped, irregularly placed cells, with branches which repeatedly divide producing a zig-zag crenated appearance ending in varicose ramifications.   These branches enmesh the spindle cells and run parallel with the cortex.

Curiously, an axis cylinder descends from these cells even going as far as the white substance. In rats, guinea-pigs and rabbits, there are no medullated fibers.

3. Zone of perpendicular spindle cells. These are arranged in three or four rows and have two polar processes, the upper and the lower. In the spindle-shaped body, the nucleus may be seen through the thin cytoplasmic layer. The upper (polar) fiber ascends to the first layer. The descending fiber reaches the fourth layer, ending in pointed bushy dendrites. After going down, this dendrite turns and ascends; at this angle of turning, an axon is given off from a dentritic expansion, as it were, some distance from the cell. This corresponds to the axon proceeding from the small pyramidal cells of other regions. It descends to the deep plexiform layer and white substance. In its descent, collaterals are given off at right angles to the parent stem, fewer the deeper the axon goes. Some spindle cells lack the ascending fiber. Cajal considers these forerunners of the fully formed bipolar cell. This third layer also possesses many star-shaped cells and descending fibers from the star-shaped cells of Zone 2 and ascending fibers from deeper lying pyramidal cells.

4. Layer deep plexiform zone. This consists of medullated and nonmedul-lated fibers running parallel to the surface. Here are found small pyramidal cells, perpendicular spindle-shaped cells (as above) and three cornered or larger fusiform elements, signalized by an axis cylinder which ascends to the first upper plexiform layer and ends in it; it gives off other collaterals to this fourth layer. Here and in the following fiber zones are three cornered, star-shaped or egg-shaped cells of larger size, whose ascending axis cylinders first descend or run horizontally and then go upward. These strong axonal expansions give off many strong long collaterals to this layer.

5. Layer of middle-sized pyramidal cells. This corresponds to the small and middle-sized pyramidal cells of other areas. The cell body is reticulated by fine pale fibers. The ascending process goes to the molecular zone: the descending fiber (axon) enters the subjacent white substance.

6. The layer of larger sized pyramidal cells. The strong apical process traverses all the layers, to end in the first layer. The axis cylinder descends to the white substance.

7. The layer of polymorphous cells contains small pyramidal cells, three cornered and spindle-shaped elements, the axons of which are lost in the white substance.

The Retrosplenial Cortex in the Mouse.—In this mammal, one can differentiate the same layers as in the rabbit. Zone 1 is markedly developed as a thick plexiform layer with many cells and a rich fiber plexus. Zone 2 contains the same type of star-shaped cells as described in the rabbit, but fewer in number. Zone 3 contains the egg-shaped cells, with two polar expansions, as in the rabbit. The axon springs from a descending dendritic stem or oftener from the cell. Processes are given off for Zone 4 and the pyramidal cell layers. Zone 4 or the plexiform layer contains many new fibers running a tangential course, which enmesh many spindle shaped and small pyramidal-shaped elements. Zones 5 and 6 are quite similar to those studied in the rabbits, but not so thick. The large pyramidal cell has an ovoid body, and sends its fiber upward to terminate in the bushy expansions of the first layer, as noted in the rabbit.

Cajal draws the following conclusions: The suboccipital or retrosplenial cortex is strikingly well developed and differentiated in the macrosomatic animals (dogs, guinea-pigs, rabbits, cats, etc.).

Its proximity to the presubiculum and Ammon's horn bespeaks a relationship to the olfactory function. Further evidence is adduced pointing to the probability of this area being a tertiary olfactory station.

BROCK, New York.

Histologic Constitution of the Pineal Gland. I. Parenchymatous Cells. P. del Rio-Hortega, Libro en honor de S. Ramón y Cajal 1:315-359, 1922.

A detailed histologic study based on observations on about fifty human pineal glands from subjects between 8 and 100 years of age. Observations on the pineal glands of several mammals and a few birds are also included. Sections were stained with the author's silver carbonate method (for the parenchymatous cells) and with Cajal's sublimate gold method (for neuroglia cells).

The pineal gland is regarded as an organ consisting of parenchymatous cells and neuroglia. The former are the most abundant. Their aspect and size are extremely variable and do not seem to be related to age. The cell body gives off numerous club-shaped or pointed processes. The processes are long and wavy, ending within the lobules of the parenchyma or in the perivascular connective tissue of the interlobular spaces. They form an intricate plexus. The cells placed near the center of the lobule appear as multipolar elements, whereas those in more peripheral zones send their processes, more or less polarized, toward the interlobular spaces. Besides the pointed and club-shaped processes there are in some cells two or three thin prolongations resembling axons (axonoid processes). The nucleus is pale, of variable shape, with scanty chromatin granules. It may show coarse masses, considered by some authors as signs of secretory activity. These masses are regarded as the result of invaginations of the nuclear membrane, already observed by Achúcarro and Sachisán, Biondi and Walter.

In addition to the structures mentioned above, the cytoplasm of the parenchymatous cells contain: (a) a delicate reticulum which shows marked affinity with silver solutions; (b) a chondrioma, represented by minute mitochondria and short chondrioconts, scattered throughout the cell body; (c) coarse pigment granules usually crowded against the nucleus; (d) spherules of different sizes resembling degenerative products, sometimes lipoids; and (e) rods or short filaments placed near the nuclear membrane. The latter appear constantly in the cells and are identical with the blepharoplasts of the ependyma cells.

Although the shape and size of the parenchymatous cells are extremely variable, the cells can be grouped under three different types. In one type, abundant in young subjects, the cells have round or kidney-shaped, eccentric nuclei and long, pointed processes. A second type is represented by the cells in the periphery of the gland, where they occur in small groups. These elements possess wavy processes which tend to surround the cell body and appear varicose in pineal glands in advanced regression. Finally, a third type is represented by elements with short, club-shaped processes as if produced by retraction. In addition to these three types, found in almost every pineal gland, there are also giant parenchymatous cells scattered through the parenchyma, but chiefly located in the periphery of the lobules. They form masses in areas deeply modified through neuroglia hyperplasia, presence of acervulus or excessive abundance of connective tissue. These cells are usually found in pineal glands from old subjects, but they also occur in the young. Their processes are irregular and end in enlargements which often surround the acervulus.

In mammals and birds, the parenchymatous cells do not show club-shaped processes. The author believes that in man these cells exhibit a marked tendency toward hypertrophy, manifested in the increase in size of the cytoplasm and the enlargement of the processes. The nature of the parenchymatous cells is discussed. Rio-Hortega believes that they are neither neuroglia cells nor nerve elements, but a specific type of the pineal gland. If the latter performs an endocrine rôle, the parenchymatous cells are not concerned in the production of internal secretion. It is suggested that the secretory elements are certain neuroglia cells with specific granulations, to be described in another contribution. As a concluding remark, the belief is expressed that the histology of the pineal gland will never be well known until it is studied with special methods during embryonic development and early infancy.

<div align="right">NONIDEZ, New York.</div>

EARLY CONVULSIONS IN EPILEPTICS AND IN OTHERS. HUGH T. PATRICK and DAVID M. LEVY, J. A. M. A. **82**:375 (Feb. 2) 1924.

The authors compare all early convulsions occurring in a group of 500 epileptic patients with those in a group of 752 "unselected" infants and children. The epileptics were private patients with idiopathic epilepsy without complications. The unselected group consisted of infants and children seen at "better baby" conferences in one urban and two rural communities in Illinois. Both groups are representative of "private practice" as contrasted with institutional and dispensary groups.

Records of early convulsions were found in ninety-eight cases, or 19.6 per cent. of the epileptic group; in the ûnselected group there was a history of infantile convulsions in thirty-two cases, or 4.2 per cent. The frequency of early convulsions in the epilepsy group was, in a general way, inversely proportional to the age at the time of the first epileptic convulsion. In their series, the time interval between infantile convulsions and epilepsy varies from several months to thirty-five years. In about 25 per cent. of cases, the interval was from ten to nineteen years, and in 15 per cent., from twenty to thirty-five years. From the frequency indicated, 4 per cent. for the nonepileptic and 20 per cent. for the epileptic group, it may be concluded that the presence of an infantile convulsion, per se, as defined in this paper, multiplies the person's chance for epilepsy later by at least five. Accepting the ordinary chances for epilepsy as one in 600, the chance of an individual with a history of infantile convulsions is somewhat less than one in 120—an incidence of some significance.

There is a preponderance of single early convulsions in the nonepileptic group (65 per cent. of the total) and of multiple convulsions in the epileptic group (76 per cent. of the total). The majority of early convulsions in the nonepileptic group (75 per cent.) occur between 6 and 17 months of age, and most of these are between 11 and 13 months. In the epileptic group, 63 per cent. of the early convulsions occur under 6 and over 17 months; only 37 per cent. between 6 and 17 months of age.

As to the type of convulsion, in the nonepileptic group the most frequent type of attack may be described as a brief generalized tonic convulsion, with little or no clonic movements, with or without fever, and without tongue biting, whereas in the epileptic group, the attacks were of longer duration and were sometimes followed by confusion or torpor; attacks limited to one limb or one side of the body are noted exclusively in the epileptic series.

Within the unselected series, the group with early convulsions contains relatively seven times as many families with instances of early convulsions in relatives as does the remainder, and a much higher frequency of epilepsy.

They conclude that convulsions in infancy and childhood not epileptic, spasmophilic or symptomatic of a gross brain lesion, are evidence in themselves of the individual's increased chance of later epilepsy and that there is no definite "interval of safety" beyond which epilepsy will not occur.

Nixon, San Francisco.

Teratogenic Tumors of the Brain (Teratoma of the Aqueduct of Sylvius). C. Jacob, Libro en honor de S. Ramón y Cajal **2**:415-431, 1922.

A description is given of a typical teratoma of the aqueduct of Sylvius in a 32 year old man with the following clinical history: The patient fell from a horse when he was a child, without suffering concussion of the brain. From the time of puberty he had complained of periodic cephalalgia with vomiting; this increased until it was paroxysmal. During the two years preceding death, dizziness increased along with diplopia and double ptosis; external divergent strabismus was also developing. At the time of examination, the patient walked with a marked tendency to fall backward. There were: paralysis of both oculomotor and trochlear nerves, nystagmus and horizontal diplopia; reflexes somewhat exaggerated; movements of the legs with slight tendency to spasm; no Babinski sign; normal sensibility; some difficulty in speech; good memory, but the mental condition was that of a child. He had no epileptiform seizures. The patient died suddenly several months after examination, from hypostatic pneumonia and degenerative myocarditis.

Inspection of the brain disclosed internal hydrocephalus with intense compression of the pontopeduncular region; the aqueduct was much enlarged and occupied by a tumor. The latter contained three types of cysts: (1) typical dermoid cysts lined with stratified epithelium and abundant sebaceous glands clearly hyperplastic in some regions, and numerous hairs with somewhat flattened follicles. Below the epidermis there was loose connective tissue with abundant smooth muscle fibers and numerous dilated blood vessels. This region appeared infiltrated with lymphocytes, eosinophils, plasma and mast cells. The contents of the cysts were similar to the contents of the cholesteatomas. (2) Cysts lined with cylindrical and low cuboidal epithelium with mucous contents, placed in the vicinity of dermoid cysts. (3) Seromucous cysts with mucous epithelium representing salivary gland tissue of the parotid type. The epithelium showed deep regressive alterations and was for the most part desquamated. The solid part of the tumor was occupied by four hyaline cartilages, one of them undergoing incipient ossification. The cartilages were united by fibrous membranes; they seemed to represent the cartilages of the larynx.

Lesions were found in the superior and inferior colliculi, central gray matter of the midbrain, oculomotor nuclei, hypothalamus, red nuclei, hypothalamic commissures and, to a less extent, in the medial and lateral fillets and the pathway of the red nuclei. Secondary degeneration of the pyramidal tract was not observed.

Jacob explains the development of the tumor by penetration of ectodermic and branchial tissue in the mesencephalon before complete closure of the latter. He suggests that the fall the patient suffered when he was a child might have

caused ependymal hemorrhage in the aqueduct, and that new vessels might have reached the enclosed embryonic tissue thus enabling its further development. The diverse syndromes observed are also discussed.

NONIDEZ, New York.

THE FINER ANATOMY OF THE PRIMARY ENDINGS OF THE NERVOUS OCTAVUS IN COENDU PREHENSILIS L. G. FUSE, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**:251-260, 1923.

The primary endings of the acoustic nerve in *Coendo prehensilis* are almost like those of the porcupine. They have in common a close relationship to the granular ganglion and the stratum granulosum of the flocculus so that no sharp division between the two exists.

There are two points of difference. The first consists in the presence of a heavy, bandlike projection from the dorsal portion of the granular area which extends mesodorsad and orally; this extends caudally as far as the caudal point of entrance of the eighth nerve and orally as far as the oral nucleus dentatus, near which it disappears. The sagittal extent is about 540 microns. The elements are strongly carminophil. The second peculiarity consists in a less differentiated granular layer of the flocculus so that in its caudal portion it lies immediately subependymal and next to the lateral recess of the fourth ventricle. In the porcupine, it is separated from the ependymal layer of this recess by a thick granular layer; only a slight indication of this structure is found in the flocculus of *Coendu*. The distribution of the acoustic fibers in the granular ganglion is similar in both animals. Small polygonal, star and spindle-shaped cells are found scattered here and their throughout the ganglion.

Fuse believed that the primary acoustic centers of the ventral ganglion may transmit the sound impulses to the higher centers directly or reflexly to the ganglion cells of other cranial nerves. Some of the impulses reaching the dorsal ganglion may be delayed and subsequently transmitted to neighboring regions of the cerebellum, such as the flocculus and the nucleus dentatus. Considered in this light, the granular ganglion acts as a giant reservoir. Why the granular ganglion should be so huge in these animals and almost absent in other mammals defies explanation, nor is it known whether other members of this family show this peculiarity.

WOLTMAN, Rochester, Minn.

THE HETEROTRANSPLANTATION (FROM ANIMAL TO MAN) OF ENDOCRINE GLANDS IN DISEASES OF THE NERVOUS SYSTEM. J. BRODSKY, J. f. Psychol. u. Neurol. **30**, Nos. 1 and 2 (July) 1923.

The author is concerned with the transplantation of parathyroid glands from animals to man ("Heteroplastik") in human cases of tetany. Making use of the investigations of Bach (Bach, A., and Zubkowa, S.: Ueber die Fermentzahlen des Blutes, Biochem. Ztschr. **125**) with regard to the katalase index of blood, and the researches of K. N. Koltzoff concerning the type of animal from a study of its blood peculiarities, Brodsky selected the goat. After the katalase index of the patient had been determined, a goat was picked from a group whose index corresponded most to that of the sick person. The author deprecates the delays in transplantation, and to obviate this, animal and patient are operated on at the same time in close proximity.

Two illustrative cases are detailed. Case 1 is that of an 18-year-old girl manifesting tetany in both upper and lower extremities, back and face. No

hereditary factors are disclosed. Her seven brothers and sister were healthy. She had had measles, chickenpox, rickets, relapsing fever and typhus. For the year prior to admittance to the hospital, tetanic seizures had been occurring in the lower extremities, especially in the toes, face, upper extremities, back and neck. Pain in the back, marked perspiration, cardiac palpitation and tachycardia (120 and over) with slight temperature elevation were noted. Menstruation had not appeared. Her Wassermann test was negative. She was completely conscious and possessed a normal psyche; cranial nerves and sensation were normal. The deep reflexes were active, with a left patellar clonus. There were marked Chvostek, Trousseau and Hoffman signs, with trismus, risus sardonicus, accoucheur hand and pes equinovarus. A deforming kyphoscoliosis developed, with a poking out of the anterior chest, making breathing increasingly difficult. To this distressing picture, decubitus was added, involving the back and gluteal regions. Other measures failing, a transplant was decided on. Accordingly, the patient's katalase index was determined according to Bach's method. It was katalase 10.2, protease 1.2, peroxydase 60, esterase 70. A goat was chosen with katalase 3.4, protease 2.3, peroxydase 50 and esterase 40. The goat and patient were operated on simultaneously. A pair of parathyroid glands was removed from the goat; one was immediately transplanted in toto into a pocket made in the patient's rectus abdominis muscle; the other was cut in two parts and likewise planted in another pocket. All trauma was minimized, and the area into which the transplant was put was kept strictly bloodless. After the operation the tetanic manifestations rapidly subsided, the kyphoscoliosis lessened, and the patient made a complete recovery. Five months later she was still in normal health.

Case 2 was that of a 26 year old unmarried woman, with painful cramps in the musculature of the hands, feet, face and neck, together with diarrhea and general weakness. Amenorrhea had existed for two months. She had had typhoid at the age of 12 and malaria at 25. There were no hereditary defects. Her urine and Wassermann tests were negative. The red blood count was 3,710,000; the white blood count was 7,450. For three months, cramps had occurred in the chest and diaphragm, accompanied with distressing attacks of suffocation. Administration of parathyroid and morphin by mouth was of no avail. Her katalase index was katalase 12.2, protease 1.53, peroxydase 60, esterase 70. A goat was selected with katalase 4.3., protease 3.2, peroxydase 50 and esterase 40. After heterotransplantation, the tetany disappeared, the menses reappeared, the bowel movements became normal, and there was a complete restitution to health.

In closing, Brodsky stresses this method of endocrine therapy, and prophesies that the future will develop it further in regard to other glandular disorders.

BROCK, New York.

FURTHER OBSERVATIONS ON PEARLY TUMORS. PERCIVAL BAILEY, Arch. Surg. 8:524 (March) 1924.

Bailey reports three more cases of pearly tumors, the "tumeurs perlées" of Cruveilhier and the "piale epidermoide" of Bostroem. He feels that "pearly tumor" is the best appellation for this type of growth. Cholesteatoma is not an exact term to use, for these tumors often do not contain cholesterin. Piale epidermoide is not satisfactory, since these tumors may be extradural and so have no connection with the pia mater.

But no matter what the nomenclature used to distinguish them may be, microscopically their appearance is unmistakable: "masses of epithelial cells of characteristic structure, which may or may not contain hair, or cholesterin crystals, and whose surface has usually the appearance of mother-of-pearl. Histologically, they are composed of a fine fibrous wall, the inner surface of which is covered by layers of flattened cells containing keratohyaline granules, and also of masses of cells flattened and stuctureless, or with only the intercellular substance remaining, giving the appearance of the cells of woody plants. The interior may be a broken down mass of débris containing fatty material and cholesterin crystals.

The favorite location for these collections of epithelial cells is in the pia mater around the base of the brain. They may project upward into the cerebellopontile angle, the fissure of Sylvius, between the frontal lobes or into the third ventricle. They often occur extradurally under the parietal or temporal bones. Rarely, they may be found in the fourth ventricle, and more frequently in the middle-ear cavity."

Pearly tumors of the middle ear do not develop necessarily from proliferation of the cells lining the cavity of the middle ear resulting from infection. It is unquestioned that the normal epithelial lining of the middle ear cavity is always replaced by squamous epithelium after long-standing perforation of the drum. "But, although the caseous residue of an old suppuration may contain cholesterin and may closely resemble the material from the interior of a pearly tumor, when it is removed, it will be found, microscopically, to be free from the peculiar woody-plant-like cells and from all trace of an epithelial sac."

The structure of granulomatous variety of cholesteatoma of the middle ear is very different from the primary tumors. "Granulomatous elements covered with stratified epithelium will be found embedded in loose lymphocytes, leukocytes, myeloblasts, and squames in all stages of fatty and granular change. The squames may be loose or grouped in closely laminated "pearls" having for the most part lost their nuclei and become strongly acid-fast. Bacteria of all kinds are mixed with the cells and the throat organisms—spirochetes, fusiform bacilli, yeasts, leptothrices, cladothrices, etc."

A primary growth in the middle ear lies for a long while dormant "or giving only negligible symptoms, but its presence predisposes the ear to infection, for which the patient first comes to the clinic. By that time, the tumor may have been involved in the inflammatory process and its original structure may be much altered. It is then believed to have been secondary to the old suppuration."

Clinical and pathologic records of three cases of pearly tumors are reviewed.

GRANT, Philadelphia.

A CONTRIBUTION TO THE KNOWLEDGE OF PANCREATIC INNERVATION. ARE THERE SPECIFIC CONDUCTORS FOR THE ISLANDS OF LANGERHANS, GLANDULAR ACINI AND VESSELS? F. DE CASTRO, Libro en honor de S. Ramón y Cajal 1:537-563. 1922.

This is a detailed study of the nerve supply of the pancreas in cats, dogs, guinea-pigs and, especially, in albinos and wild mice. The methods of Golgi (double impregnation of Cajal) and Cajal (reduced silver nitrate) were used in most of the cases. A few preparations were stained with Ehrlich's methylene blue and Spielmeyer's stain for myelin.

The pancreas receives medullated and nonmedullated nerve fibers, forming bundles in contact with the vessels, thus forming a perivascular plexus. The acini are supplied by nonmedullated fibers which form periacinose plexuses; the branches of these plexuses end between or around the epithelial cells. These nonmedullated fibers are independent of those reaching the islands and the vessels. The islands receive medullated and nonmedullated fibers which form rich peri-insular plexuses. They arrive at the islands along with the vessels or as isolated bundles; the latter do not participate in the innervation of the acini, running between them without giving off collaterals. The nonmedullated fibers of the peri-insular plexuses end in the periphery of the island or penetrate . among the cell cords, branching and sending short prolongations which end in contact with the insular cells. The medullated fibers, on the contrary, pass over the surface of the island without sending collaterals to its cells. Some of these fibers end in microsympathetic ganglia, and others send collaterals to the vessels.

The large and middle-sized vessels of the pancreas possess three nerve plexuses, namely, adventitial intermediate and muscular. These plexuses are formed by nonmedullated fibers and collaterals coming from the perivascular bundles. In small vessels, there are only adventitial and muscular plexuses. In addition to these nonmedullated plexuses, the large vessels also receive arborizations from medullated fibers, exhibiting for the most part a longitudinal orientation.

In the pancreas of the mouse there is a rather large sympathetic ganglion made up of multipolar cells surrounded by a plexus formed by arborization of large medullated fibers. The preganglionic bundles consist of medullated and nonmedullated fibers; the postganglionic are increased with the axons of the ganglion cells. There are also microsympathetic ganglia in contact with the islands and vessels; they consist of large and small multipolar cells and are connected by nonmedullated fibers forming intricate plexuses.

The conclusion is reached that the acini and islands of Langerhans are exclusively supplied by nonmedullated fibers, which are, however, independent to the extent that in no case do the acini receive branches from the fibers which supply the islands, or vice-versa. Furthermore, there is absolute independence between the nonmedullated fibers supplying the blood vessels and those ending in the glandular portion of the organ.

NONIDEZ, New York.

THE SIGNIFICANCE OF AIR INJECTIONS IN CEREBRAL AND SPINAL CORD DIAGNOSIS. W. WEIGELDT, Deutsch. Ztschr. f. Nervenheilk. **77**:165 (March) 1923.

Dandy's first publication on air injections into the ventricles appeared in July, 1918. In October, 1919, he reported the cases of eight patients in whom the intraspinal route had been employed, but because of unsatisfactory results he did not recommend this method. In 1920, Bingel and Wideröe simultaneously published two articles on the intraspinal method. The author reports sixty-five cases with 104 air injections by the intraventricular (Dandy) and intraspinal method (Bingel). Unpleasant manifestations were less frequent with Dandy's method. Objections to Dandy's method were: (1) a more serious procedure (hemorrhage; injury to brain tissue), (2) difficulty in filling ventricles when lateral ventricles are narrow or in abnormal position because of tumor mass; (3) local anesthesia, and (4) a more extensive instrumentarium. Bingel's method is simpler and requires fewer instruments; no serious reactions were observed in the author's series. A slight collapse occurred in one case. The

most frequent complaints were severe headaches, usually frontal, nausea and vomiting, a rushing or boiling sensation following Bingel's method. These symptoms usually subsided within from two to four hours in the recumbent posture. No cellular increase in the spinal fluid, as noted by Hermann in three cases, was observed. Ventriculography is a valuable diagnostic aid, especially in laterally located tumors.

No results were obtained in migraine. In epilepsy, the convulsions became less frequent following injections of air, but only in one case remained absent longer than one month. The absorption time of the injected air was studied by serial roentgen-ray examinations. In normal cases, the air was absorbed from the subarachnoid space in from three to five hours and from the ventricles in from six to ten hours. In markedly dilated ventricles, air was noted after from two to three weeks and in one case after four weeks.

A complete occlusion of the spinal canal in cord lesions (fourteen cases, tumor, caries, etc.) was not observed in a single case. In five cases, a partial occlusion was noted; below the level of the lesion, xanthochromia was found, while above, the spinal fluid was normal.

In all fourteen cases, the patient complained of marked shooting pains at the level of the compression, especially when the air pressure was altered. This is an important localizing sign.

HAMMES, St. Paul.

SHOCK TREATMENTS IN MENTAL DISEASES. C. PASCAL, Presse méd. **32**:234 (March 15) 1924.

Results are analyzed in a number of series of mental cases, personally observed by this investigator, in which different types of "shock," or reaction, were brought about by the hypodermic administration of different substances.

Little benefit was observed after injections of vaccines. Ordinary horse serum was valueless. Diphtheria serum helped in one case only. Autogenous cerebrospinal fluid similarly benefited but one patient in the series.

Colloidal gold and silver injections were sometimes beneficial; also milk; sodium nucleinate, in doses of 0.2 to 0.4 gm., occasionally relieved symptoms. Arsphenamin was helpful chiefly in patients with general paralysis, less so in other syphilitic psychoses (here the author does not make it clear whether a toxic preparation, causing as it does a more severe shock of reaction, would not be better than a "nontoxic" one).

Turpentine abscesses seemed to be the best form of shock tried. One cubic centimeter was not too painful; it caused a sudden rise in temperature after twelve or fourteen hours, which lasted about three days; after twelve days, no local traces remained. Five-tenths cubic centimeter was given in some cases, and repeated several times at weekly intervals; this amount caused less local and quite as much general reaction. Turpentine injections quieted psychomotor unrest, and removed negativistic symptoms. Manic depressive and epileptic manias improved more than catatonic excitement. In excited paranoid cases, the excitement was quieted and the paranoia unaffected. Depression was less benefited. Malingerers were betrayed—this is a rare but valuable use for the treatment.

The shock of gross physical injury—fractures, etc.—brought no consistently good results in the few cases observed. Similarly with the reactions from intercurrent diseases, as observed in epidemics of erysipelas, pneumonia and dysentery; if there was any alleviation of mental symptoms, it was only temporary.

Most of all, the method as a whole required further study of dosages. Turpentine abscesses were best. Some form of shock was the treatment of choice in violent and refractory agitations.

HUDDLESON, New York.

EXPERIMENTAL CHOREA AND ATHETOSIS (PRELIMINARY NOTE). G. R. LAFORA, Libro en honor de S. Ramón y Cajal, Madrid 2:261-263, 1922.

Lafora has operated on twenty-seven cats, producing the choreic or the athetotic syndrome in twelve cases. Lesions were produced: (1) in the red nucleus; (2) in the hypothalamus, and (3) in the superior cerebellar peduncle and red nucleus. The syndromes obtained were: monoplegic chorea, hemichorea, chorea of the four limbs, athetosis of the four limbs, and intentional, monoplegic and hemilateral spasms. In some animals, there were more or less transitory cerebellar syndromes, in others hemianopsia, nystagmus or ophthalmoplegia. The syndromes were in some cases homolateral, in others contralateral. The involuntary motor syndrome appears sometimes a few hours after the operation, disappearing within two or three days; in other animals, it appears a month after the operation and lasts several months. Two types of choreic movements were observed: some were rhythmic, sudden, of myoclonic type, while others were more complex and more similar to the wide movements of human chorea.

With regard to the localization of the lesion and its effects, the author reaches the following conclusions: 1. A lesion of the superior cerebellar peduncle constantly produces homolateral or contralateral chorea, according to the more or less anterior position of the lesion; this form corresponds to the *Bindearmchorea* of Bonhoeffer. 2. A hypothalamic lesion seems to produce with certain constancy the athetotic syndrome, which may be bilateral when the lesion crosses obliquely the midline injuring both hypothalami. 3. A lesion of the red nucleus or the rubrothalamic pathway between the latter and the hypothalamus produces mixed choreic and athetotic syndromes. These results seem to favor in some respects Kleist's theory on the localization of the different extrapyramidal motor syndromes. Lesions of both lenticular nuclei were not followed by any choreo-athetotic syndrome.

NONIDEZ, New York.

GIANT NERVE FIBERS IN CRUSTACEANS, WITH SPECIAL REFERENCE TO CAMBARUS AND PALAEMONETES. G. E. JOHNSON, J. Comp. Neurol. 36:323, 1924.

In the ventral nerve cord of the crustaceans studied, three sets of nerve fibers of giant dimensions were found: (1) a median set which terminates anteriorly in the brain where the tract decussates; (2) an intermediate set of motor fibers which is present in each third pair of nerves of the abdominal ganglions and whose cells of origin lie in the opposite half of the ganglion anterior to the nerve with which it is connected, and (3) a lateral segmental set in each thoracic and abdominal ganglion. The latter giant fibers are not continuous throughout the cord, but are formed in each segment by the union of two small branches, one from each side of the ventral nerve cord. Traced anteriorly from its ganglion of origin, a fiber of this system passes through the next ganglion and terminates in contact with the side of the next anterior segmental giant fiber. Although this relation in *Cambarus* is one of contact with short branches, a difference in appearance of the two fibers is present at the region where they are most intimate. The fact that degeneration did not extend from one segmental giant fiber into another also supports a contact conception of this neuronic connection. In *Crangon*, the fibers are separated

only by a clear space; the cytoplasm is continuous, but the neurofibrillae are separate. In *Polaemonetes,* there is evidence that the neurofibrillae of one fiber extend into the cytoplasm of the other, but there is no evidence that the neurofibrillae are continuous. In these crustaceans, three types of neuronic connection are demonstrated; namely, by contact with an intervening membrane, by contact without an intervening membrane, and by extension of the neurofibrillae of one fiber a short distance into the axis cylinder of the other.

GRAY, Chicago.

VISCERO-CUTANEOUS ANAEMIC ZONES AND THEIR SIGNIFICANCE. T. B. WERNOE, J. Neurol. & Psychopath. **4**:103, 1923.

By exposing the skin of patients suffering with visceral disease to cold air, a localized mild ischemia becomes visible in a subdued light, the limits of which correspond accurately with the zone of cutaneous hyperalgesia. The author has examined about 3,000 patients by this method since the spring of 1920; fifty of these cases have been confirmed as to diagnosis by operation, necropsy or roentgenology. As the result of these observations, the author concludes that the results recorded by Head, Faber, and Mackenzie from studies of hyperalgesia alone are inaccurate. Similar results have been secured experimentally by the local action of epinephrin on the gallbladder, spleen, liver and intestine, and also in cold-blooded animals from faradic stimulation of the intestine. The zone of ischemia and hyperalgesia is always fan shaped, with the base directed toward the midline, and the localization of the afferent segmental innervation of the various organs has been carefully mapped out and is found to be constant. The author gives the following general rules relating to the occurrence of the reflex hyperalgesia: (1) Reflex hyperalgesia originating in *unpaired* organs is always *bilateral.* (2) *Unilateral* reflex hyperalgesia is due to disease of *paired* organs. (3) The organs have the same afferent innervation as the vessels supplying them. (4) The lower border of the reflex hyperalgesia in cases of obstruction of a lumen corresponds to the afferent segmental innervation of that part of the organ which lies immediately proximal to the obstruction.

SINGER, Chicago.

ON THE DIFFICULTY OF UTILIZING APHASIC SYMPTOMS IN THE LOCALIZATION OF BRAIN TUMORS: WITH A REPORT OF FOUR CASES WITH NECROPSY. I. S. WECHSLER, J. Nerv. & Ment. Dis. **59**:31 (Jan.) 1924.

The author reports four cases of brain tumor presenting aphasic symptoms which were either misleading or of questionable value in the localization of the lesions. The patient in Case 1 presented practically a "pure" motor aphasia with right hemiparesis and astereognosis. The tumor did not involve Broca's area, but was situated deeply in the right (?) post-central area in the parietal lobe extending into the lateral ventricle. The patient in Case 2 presented a right hemiparesis, doubtful astereognosis, difficulty in carrying out commands, "word-naming aphasia" and marked choked disks. Necropsy showed a hemorrhagic glioma located entirely in the frontal lobe. The patient in Case 3 presented a right hemiplegia with difficulty in naming objects and finding correct words to express himself, suggesting a motor aphasia. Necropsy revealed a glioma of the temporosphenoidal lobe, extending into the occipital but not into the frontal lobe. The patient in Case 4 had been left-handed since birth and had jacksonian attacks on the left side. She could not speak,

but she heard and understood spoken words. Both motor and Broca's area appeared to be involved. The right frontal region was explored surgically; no tumor was found. Necropsy revealed a glioma springing from the right frontal region but occupying principally the space between the two frontal poles and infiltrating the left frontal lobe and corpus callosum. Wechsler accordingly believes it doubtful whether one can speak of circumscribed speech centers or localize tumors by means of aphasia alone.

VONDERAHE, Cincinnati.

ALTERATION OF THE RETICULUM OF GOLGI IN RABIES. J. RAMÓN Y FAÑANÁS, Libro en honor de S. Ramón y Cajal **1**:565-578, 1922.

This is a study of the alterations of the reticulum of Golgi in nerve cells of dogs having died of rabies. The hippocampus, cerebellum, spinal ganglia, gasserian ganglion and ganglia of the vagus were examined. Cajal's urano-formaldehyd method was used for the impregnation of the reticulum.

According to the author, the reticulum of Golgi is perhaps the first part of the nerve cell to show deep alterations in rabies. In all his researches, he has not been able to find a single cell exhibiting a normal reticulum, even in those elements which appear normal in sections stained with anilin dyes. The lesions of the reticulum are most marked in the sensory ganglia, especially in the gasserian and plexiform of the vagus. Fañanás has also found deep alterations in the spinal ganglia.

The changes in the reticulum are caused by its fragmentation, accompanied by weaker impregnation of the argentophil material. The trabeculae become independent, acquire a granular aspect, and either persist in a restricted portion of the protoplasm or are scattered throughout the cell body, sometimes occupying the periphery of the cell. In cases of advanced fragmentation the reticulum appears as small granular masses, granular rings, club-shaped rods, or rods with much curved ends simulating a terminal ring. In extreme cases of destruction the protoplasm may appear occupied by small, pale rods and rings. The latter may exhibit a marked tendency toward hypertrophy. The alterations in the pyramidal cells of the hippocampus are not so marked as in the other parts examined and do not seem to be specific of rabies.

NONIDEZ, New York.

SOME NOTES ON THE DOUBLE INNERVATION OF MESODERMAL MUSCLE. R. A. DART, J. Comp. Neurol. **36**:441, 1924.

The unisegmental striated muscles of the ventral body wall of the python have been demonstrated to possess, in addition to the usual somatic nerves which are coarsely myelinated, two types of nerve fibers which are presumably of sympathetic origin; of these two types, one is absolutely unmyelinated and ends in a bulbous expansion on the sheath of the individual muscle fibers, while the other is finely myelinated and terminates below the sarcolemma in bulbs, the arrangement of which resembles somatic motor end-plates. The unmyelinated type of sympathetic fiber is thought to be sensory, and the very finely myelinated type to be motor. There is no anastomosis between the sympathetic and somatic fibers to a muscle, but the former intermingle with each other and are distributed in a plexiform manner. In the perimysium, there are mixed nerve trunks exhibiting ganglionic masses in addition to nerve fibers. These ganglia may possibly be the source of the sympathetic fibers to the striated muscles. They are probably analogous to the sympathetic ganglia in the viscera.

Every muscle spindle was found to possess one or more finely medullated fibers, the character of whose terminations indicated that they were sympathetic motor fibers. Such sympathetic motor fibers to muscle spindles may explain the resistance of the muscle spindles to atrophy after section of the somatic motor nerve to that muscle.

Dart suggests that the sympathetic motor and sensory elements described may be links in a reflex mechanism which may possibly subserve reflex vasodilatation following muscular contraction. They may also play some rôle in the perception of muscle sense and the maintenance of muscle tone.

GRAY, Chicago.

BLOOD AND NERVE AS CONTROLLING AGENTS IN THE MOVEMENTS OF MELANOPHORES. LELAND C. WYMAN, J. Exper. Zool. **39**:73 (Feb. 20) 1924.

By making a vertical incision through a part of the tail of a specimen of *Fundulus heteroclitus L.*, the pigment motor nerves to the melanophores in the region posterior to the incision are severed and the distal portions of these nerves degenerate in a few days. The blood supply to this region remains normal. The melanophores in the denervated area thus produced expand at first, but in four or five hours they partially contract, assuming a stellate form, and remain in that condition until acted on by a direct stimulus. These denervated melanophores can be used as indicators to determine whether chemical substances introduced into the body of a fundulus at a point anterior to the incision have their action directly on the pigment cells being carried to them by the blood or have an indirect action through the nervous system. By this method, it is found that a number of anesthetics and alkaloids when applied to the gills or injected into the body cavity cause various responses of the melanophores due to the action of the chemicals on some part of the nervous mechanism. Direct application of the same substances to the denervated melanophores causes similar responses unless the chemical is one which rapidly kills the protoplasm of the cells. Alcohol, ergot and several endocrine secretions when applied to the animal in the same way act directly on the melanophores being carried to them in the blood. Salts have no indirect effect on melanophores. Although there is a close relation between the melanophores and the sympathetic nervous system, the cells can react normally when freed from nervous impulses. Direct control by hormones or other chemical substances in the environment is an important adjunct to nervous control.

WYMAN, Boston.

A CASE OF UNILATERAL BULBAR LESION, PROBABLY SYRINGOBULBIA, WITH SPECIAL REFERENCE TO THE SENSORY PATHWAYS IN THE MEDULLA. W. G. WYLLIE, J. Neurol. & Psychopath. **4**:148, 1923.

A girl, aged 16, had symptoms that developed slowly and progressively. The principal points in the neurologic condition were: loss of the corneal, palatal and pharyngeal reflexes; good palatal movement but fixation of the left vocal cord in phonation; deviation of the tongue to the left; no affection of the ocular nerves except that the left pupil and palpebral fissure were narrowed; no involvement of the seventh, eighth or eleventh cranial nerves; practically no motor change in the body and limbs, with the exception of slight left asynergia; crossed hemianesthesia (left side of face and right side of neck and parts below) with dissociation. There were: faint loss of tactile

sensibility; complete analgesia on the left side of the face, partial analgesia of the right side of the body; complete loss of sensibility to cold, partial loss to heat; practically no loss of compass point discrimination, weight estimation, stereognosis, sense of vibration; no loss of sense of position or passive motion; pressure pain sense definitely but mildly diminished. The lesion was considered to be most probably a syringobulbia in the left half of the medulla, with its greatest dimension at the level of the olive.

SINGER, Chicago.

A PLEA FOR AN IMPROVED STEEL HELMET IN THE NEXT WAR. ADOLPH M. HANSON, Mil. Surgeon **53**:608 (Dec.) 1923.

The author points out the defect in steel helmets now in use in the American, French and British armies, showing that, although the cerebrum is afforded more protection than is given by the German helmets, the cerebellum remains completely exposed. An analysis made by Sargent and Smith, from cases in the British army, shows that the mortality of patients without helmets was almost twice as great as those injured while wearing helmets.

The tables show many cerebellum injuries occurring in cases when helmets were worn. The author points out the relatively small percentage of head injuries in the German reports, being a little over 3 per cent. of wounds, whereas in the allied armies, head injuries constitute 10 per cent. of all wounds. He attributes this difference to the superiority in type and construction of their helmets, and suggests a new model adapted to protect adequately the vital centers about the cerebellum.

TEMPLE FAY, Philadelphia.

VARIATIONS IN THE BABINSKI REFLEX. A. REMUS, Deutsch. Ztschr. f. Nervenheilk. **79**:366 (Sept.) 1923.

Sahli, in his textbook, states that a positive Babinski sign can be produced by various irritations of different areas of the body, such as downward pressure of the patella, pressure on the intercostal nerves, etc. This phenomena is always associated with an increase of the other reflexes.

Auerbach reports the case of a 9 year old boy, who had a depressed skull fracture of the midparietal region, producing a lesion in both paracentral convolutions. Irritation of the skin anywhere on the lower extremities and of the abdomen up to the level of the umbilicus produced a positive Babinski sign. The author reports three cases of epidemic encephalitis in which the Babinski sign was negative when tested in the usual way, but strongly positive when the skin in the mid-portion of the inner surface of the thigh (second lumbar)

was irritated by pinching. In one case, the Gordon sign also was positive. In a case of influenza with meningitis serosa, a similar phenomena was observed. In another case of influenza with marked petechial hemorrhages, severe right-sided headache and muscular twitchings of the left side of the face, arm and leg, this second lumbar positive Babinski sign was noted. In other conditions, such as multiple sclerosis, tabes and cerebrospinal syphilis, it was negative. But in one case of syphilitic triplegia it was found positive on one side, with an ordinary positive Babinski sign. The author believes that this method of second lumbar skin segment irritation for testing for the Babinski sign is more sensitive than the usual procedure.

HAMMES, St. Paul.

EXPERIMENTAL STUDIES INDICATING AN INFECTIOUS ETIOLOGY OF SPASMODIC TORTICOLLIS. EDWARD C. ROSENOW, J. Nerv. & Ment. Dis. **59**:1 (Jan.) 1924.

Rosenow questions the view that spasmodic torticollis is a functional neurosis. Working with micro-organisms obtained from infected areas of the nasopharynx, teeth and tonsils injected intracerebrally, subdurally and intravenously into rabbits, the author finds evidence of specific localizing properties manifested in the organisms. Injected rabbits developed abnormal movements of the head, and at necropsy the brains revealed perivascular infiltration in certain areas of the brain.

The spontaneous appearance of leukocytic and mononuclear infiltration about blood vessels in the brains of rabbits has been recently noted in 40 per cent. of supposedly normal rabbits by McCartney (*J. Exper. Med.* **39**:51 [Jan.] 1924). These lesions represent the consistent pathologic findings in Rosenow's rabbits; in the rabbits, however, that had recovered and were anesthetized, the actual demonstration of the streptococci was difficult or impossible. Rosenow's control animals apparently showed no perivascular infiltration. The author could find no parallelism between the degree of symptoms and the extent or exact location to microscopic lesions.

VONDERAHE, Cincinnati

DISTRIBUTION OF THE FIBERS ORIGINATING FROM THE DIFFERENT BASAL CEREBELLAR NUCLEI. W. F. ALLEN, J. Comp. Neurol. **36**:399, 1924.

The deep nuclei of the cerebellum of the guinea-pig are arranged in two sets: a lateral one, including the nucleus dentatus and the nucleus intermedius (nucleus globosus plus nucleus emboliformis), and a medial one representing the nucleus fastigii. The fibers of the brachium conjunctivum originate solely from the lateral set of nuclei. After decussation, they separate into a main cephalic bundle and a minor caudal bundle. The former sends fibers into the nucleus ruber, zona incerta, and subthalamic region and terminates in the median portion of the ventral nucleus of the thalamus. The caudal bundle is distributed to the reticular formation in the pons region. The afferent supply of the lateral set of deep nuclei is from the cortex of the cerebellar hemispheres.

The nucleus fastigii gives origin to the cerebellobulbar fibers chiefly on the same side but also to some on the opposite site. These fibers terminate chiefly in the lateral vestibular nucleus and the adjacent reticular formation. A few cerebellobulbar fibers were seen to enter the medial longitudinal fasciculi and pass to the nucleus of the oculomotor nerve. The afferent supply of the nucleus fastigii is through direct vestibular root fibers, fibers from the vestibular nuclei, and from the vermis. It may also receive some spinocerebellar fibers.

GRAY, Chicago.

DECEREBRATE PHENOMENA IN MAN. WALTER FREEMAN, L'Encephale **19**:91 (Feb.) 1924.

This paper is a brief analysis and classification of the various types of phenomena which may be regarded as evidence of "decerebration," a term which implies altered function of certain centers on either a physiologic or anatomic basis.

Some of the centers which may, through disease or altered function, produce decerebrate phenomena, are the bulbomedullary, pontobulbar, mesencephalic and cortical.

"Decerebration" may be physiologic and temporary, e. g., the normal epileptoid reaction, states of loss of consciousness accompanied by certain motor phenomena (Stokes-Adams syndrome) and intracranial hypertension. Anatomic decerebration occurs clinically in partial form in cerebral hemiplegia, cerebral scleroses and in lesions cutting off cortical control over lower centers. In complete form, anatomic decerebration is observed in the terminal stage of cerebral trauma, intraventricular hemorrhage and in certain encephalopathies.

HYSLOP, New York.

THE NERVOUS CHILD. R. G. GORDON, J. Neurol. & Psychopath. **4**:125, 1923.

In the study of why a child behaves in a way that is not normal, an answer is required to three questions: (1) What initial equipment did he start with—what was his predisposition? (2) What has been and is the influence on his mind of his bodily functions, the secretions of his ductless glands, the efficiency of his digestion, renal function and so on? (3) What have been and are the outside events which constitute his experience? The author discusses these questions in the light of McDougall's instinctive dispositions, and suggests that it is of value to consider the child as possessing certain proportions of these dispositions, although he admits that we have at present no means of evaluating them in a quantitative way. This, however, is not sufficient since, as has been pointed out by Lloyd Morgan, it is necessary to consider also the character of the integration of these various elements. Failure in this process of integration is what constitutes the nervous child and this indicates "something the matter with the frontal lobes." In certain cases "there is something inherent. . . . What that is we have not the slightest idea. In other cases nutritive deficiencies, lack of vitamines, etc., seem to be responsible." Finally lack of integration may be the result of circumstances in the environment, a factor that must never be lost sight of.

SINGER, Chicago.

VENTRAL AND DORSAL IMPLANTATIONS OF THE LIMB BUD IN AMBLYSTOMA PUNCTATUM. J. S. NICHOLAS, J. Exper. Zool. **39**:27 (Feb. 20) 1924.

A limb bud transplanted to the dorsal or ventral midline has the potency for the production of two limbs. The two limbs are symmetrical with each other and each corresponds to the side on which it develops when transplanted with normal relationship to the anteroposterior axis of the body. It is reversed in asymmetry when the relationship is reversed. The dorsal and ventral midlines are distinctly unfavorable locations for the limb. In the majority of cases, development of the limb is initiated in the new location, but complete development does not take place. When the development proceeds, the limbs are topographically complete in every particular and contain nerves

which are derived from the nearest nerve trunks. The ventrally implanted limbs get their innervation from nerve trunks which go to the ventrolateral musculature, while the dorsally implanted limbs secure innervation from the segment of the cord in proximity to them. No hyperplasia of the sensory or motor elements of the cord has been noted in connection with the ventral implantations. In the case of the dorsal implantations, however, there is hyperplasia of the dorsal region of the cord. This hyperplasia is not found in any animals in which there is incomplete limb development. Resorption is the greatest factor in the failure of the limb to grow properly. This is probably caused by lack of nutrition resulting from an inadequate blood supply.

WYMAN, Boston.

THE MOTOR CORTEX OF THE OPOSSUM. P. A. GRAY, JR., and E. L. TURNER, J. Comp. Neurol. **36**:375, 1924.

An extensive motor center for fore leg movements was mapped out on the dorsolateral surface of the hemisphere behind the orbital fissure which bounds the frontal lobe posteriorly. A smaller center for tongue and jaw movements was found at the lateral extremity of the orbital fissure. Snout movements followed stimulation on the rostral lip of the orbital fissure at about its midpoint and closure of the orbicularis oculi muscle from stimulation in the center of the frontal lobe. Ear movements were obtained from a region in the posterior inferior parietal portion of the hemisphere. The movements mentioned above were crossed. Bilateral movements of the vibrissae could be elicited from a crescentic area in front of and below the orbital fissure. No center was found for hind leg or tail movements.

Extirpation of the electrically excitable area did not produce any permanent motor defects. Within this area several histologically distinct areas can be identified. In contrast to the situation usually seen in higher mammals, the greater part of the motor area of the opossum is characterized by a granular type of cortex.

GRAY, Chicago.

SPINAL FLUID IN BRAIN TUMOR AND SEROUS MENINGITIS. OTTORINE BALDUZZI, L'Encephale **19**:83 (Feb.) 1924.

The author says that a study of the cerebrospinal fluid may be useful in differentiating between brain tumor and serous meningitis. He refers to the work of Ayala.

Increase of tension is common in both conditions. The residual pressure after withdrawal of a given quantity of fluid depends not only on the initial pressure but on the total quantity of cerebrospinal fluid in the subarachnoid spaces.

By recording the initial pressure, the amount of cerebrospinal fluid drawn and the final pressure, one may calculate a "spinal fluid quotient." The final pressure should be the average normal pressure for the position of the body in which the puncture is made.

The equation is as follows:

$$\frac{\text{Amount of spinal fluid drawn} \times \text{final pressure}}{\text{Initial pressure}} = \text{cerebrospinal fluid quotient.}$$

Ordinarily, in brain tumor, the total amount of cerebrospinal fluid present is not increased. The quotient of brain tumor, therefore, is low and varies between 2.5 and 4.5. In serous meningitis, however, the total amount of cerebro-

spinal fluid is increased, and the quotient is accordingly high, averaging between 7 and 10. When a tumor is accompanied by hydrocephalus, as in posterior fossa growths, the quotient may deceive.

Balduzzi maintains that in general the quotient is of distinct value in differential diagnosis.

HYSLOP, New York.

THE RÔLE OF TRAUMA IN THE ETIOLOGY OF ORGANIC AND FUNCTIONAL NERVOUS DISEASE. S. A. KINNIER WILSON, J. Neurol. & Psychopath. **4**:133, 1923.

Viewed in the light of the effect of the serious injuries and accidents of war experience, the claims that trauma is a causative factor in the production of organic nervous diseases of the types of disseminated sclerosis, cerebral tumors, and neurosyphilis, must be considered utterly without foundation. Similarly with regard to epilepsy; in the author's experience 80 per cent. of the cases of post-traumatic epilepsy presented definite evidence of a neuropathic predisposition in the family history or individual make-up of the patients. With regard to the relation of trauma to functional disorders, the facts are more difficult to establish; nevertheless, the mounting frequency of traumatic neurasthenia, miners' nystagmus, and other conditions, with the passage of the Workmen's Compensation Acts in England, lead to the conclusion that factors other than the trauma are of far greater significance in the evocation of the disorder.

SINGER, Chicago.

DOES BISMUTH PASS INTO THE CEREBROSPINAL FLUID, AFTER BEING INJECTED INTRAMUSCULARLY OR INTRAVENOUSLY? E. JEANSELME, M. DELALANDE and TERRIS, Presse méd. **32**:245 (March 19) 1924.

After referring to the positive results claimed by Fournier and Guenot in 1921, and Demelin and Dessert in 1922, these authors conclude from their own experiments that bismuth cannot be recovered from the cerebrospinal fluid after injection into the blood or the muscles, whether the meninges be normal or diseased.

The method used was the Aubry modification of Léger's. The reaction is supposed to detect one part in 100,000. Several sources of error are explained, which will tend to give quasipositive results when bismuth is not actually present in a test solution.

The bismuth salts administered clinically included sodium and potassium tartrobismuthate, quinin iodobismuthate, bismuth hydroxid and others. Types of cases included acute and chronic syphilitic meningitis, tabes, general paralysis and systemic syphilis without meningeal signs.

HUDDLESON, New York.

REACTIONS OF HYDRA TO CHLORETONE. W. A. KEPNER and D. L. HOPKINS, J. Exper. Zool. **38**:437 (Jan. 5) 1924.

Chloretone is a specific poison for neurons. A hydra, under the influence of this drug, loses the power to operate those muscles that are stimulated only through nervous tissue, while those muscles that are stimulated directly, retain their power to contract. In reacting to chloretone, the ectoderm behaves as a neuromuscular tissue; while the endoderm functions as a neuroid tissue. A sphincter at the base of each tentacle prevents food from passing from the enteron into the tentacle, but does not prevent the passage of material from the tentacle into the enteron. There is no extensive diffusion of absorbed

chloretone through the tissues of the body. A diploblastic animal, therefore, cannot possess anything comparable to a circulatory medium. Green hydras under ordinary conditions withstand the effects of chloretone much more than do brown and gray ones. The experiments indicate that the presence of zoochlorellae in *Hydra viridis* are responsible for its resistance to chloretone, for this resistance can be greatly lowered by placing the green hydra in the dark. This lends weight to the contention that the algae of the green hydra's endoderm are symbionts.

WYMAN, Boston.

COMPARATIVE RESULTS OF COLLOIDAL GOLD AND COLLOIDAL MASTIC TESTS. HARRY WASSERMANN, Arch. Int. Med. **33**:401 (March) 1924.

The author compared the results in 1,707 spinal fluids subjected to (1) the colloidal gold reaction of Lange and (2) the colloidal mastic reaction. The latter test was carried out with a solution of gum mastic following the technic of Stanton (ARCH. NEUROL. AND PSYCHIAT. **4**:301 [Sept.] 1920). In 87.7 per cent., there was agreement between the two tests; in the 12.3 per cent. in which there was disagreement, the colloidal mastic test was more often positive than the gold test. A paretic type of curve occurred more frequently with the mastic reaction than with the gold reaction; the prognostic value of the mastic reaction as an indication of paresis is accordingly of less value than the colloidal gold reaction. The author thus prefers to speak of abnormal colloidal curves," and points out that a paretic type of curve is not necessarily indicative of paresis, although it occurs most frequently with this disease. The author recommends the wider use of the colloidal mastic test because of the simplicity of the preparation of reagents and because its delicacy is equal to or slightly greater than that of the gold test.

VONDERAHE, Cincinnati.

FURTHER STUDIES ON INHERITANCE OF EYE DEFECTS INDUCED IN RABBITS. M. F. GUYER and E. A. SMITH, J. Exper. Zool. **38**:449 (Jan. 5) 1924.

Details are given concerning the production and inheritance of eye defects in two different strains of rabbits. In one strain the anomalies were induced in the fetal young by injecting into the mother a foreign serum immunized to rabbit lens; in the other strain, similar defects were secured in the unborn young by direct injection of pulped lens into the pregnant mother. Pedigree charts of typical matings are included and discussed. Reasons are advanced for believing the modifications to be a specific immunologic effect, and additional charts and descriptions of male line extractions showing that the hereditary nature of the defects is unquestionable, are presented. That germinal constitution can be altered by immunologic influences, the experimenters believe is the best working hypothesis to account for their results as they stand at present.

WYMAN, Boston.

A CASE ILLUSTRATING THE ETIOLOGY OF THE ARGYLL ROBERTSON PUPIL. F. J. NATTRASS, J. Neurol. & Psychopath. **4**:162, 1923.

The case described is that of a shrapnel wound of the head in which, from roentgenograms, it is demonstrated that the bullet must have traversed the midbrain and probably damaged both superior quadrigeminal bodies. The injury resulted in bilateral Argyll Robertson pupil and defect in conjugate upward

movement of the eyes. In addition, it produced right hemitremor, involving the face, arm and leg. The Wassermann reaction of the blood was negative, and there was no other evidence of nervous syphilis.

SINGER, Chicago.

THE PYRAMIDAL TRACT OF THE VIRGINIAN OPOSSUM (DIDELPHYS VIRGINIANA). E. L. TURNER, J. Comp. Neurol. **36**:387, 1924.

The pyramidal tract was followed by the Marchi degeneration method in three specimens. It occupies the usual position in the upper part of its course. At the lower levels of the medulla, there is a complete decussation into the dorsal funiculi of the opposite side. No degenerated fibers were found below this decussation. Since Cajal and pyridine-silver sections furnished no evidence of unmyelinated fibers below the decussation, it is thought that the pyramidal tract synapses at this point. The failure of the pyramidal tract to descend into the lower levels of the cord may explain the absence of any motor center in the cortex of the opossum controlling hind leg movements.

GRAY, Chicago.

Book Reviews

TEXTBOOK OF PSYCHIATRY. By Prof. DR. EUGEN BLEULER, Director of the Psychiatric Clinic, Zürich. Authorized English Edition by A. A. BRILL, PH.B., M.D., former Assistant Physician of the Central Islip State Hospital and Assistenz-Arzt of the Clinic of Psychiatry, Zürich; Lecturer on Psychoanalysis and Abnormal Psychology, New York University. Cloth. Pp. 635. New York: The Macmillan Company, 1924.

This translation has been made from the fourth German edition, the first edition having appeared in 1916. It is intended primarily for students and general practitioners, and presents in an admirably clear manner the established facts of psychiatry. In describing the different named mental diseases, the author follows closely the classification of Kräpelin, although he refuses to accept some of the finer subdivisions made by that author. The formal descriptions are well exemplified by illustrative cases in which the psychologic mechanisms involved are pointed out. These interpretations are kept strictly within the limits of clear demonstrability, and carefully avoid speculation. The author pays full tribute to the contributions of Freud, but does not follow him slavishly. The use that is made of these formulations not only will not offend those to whom psychanalysis, as often expounded, is objectionable, but will help to bring a clearer understanding of the practical value of Freud's work on psychopathology. Bleuler is equally conservative and discriminating in his discussions of pathologic anatomy and endocrinology.

The introductory chapters on psychology and psychopathology will be found of especial value. They are eminently practical, interpretative and illuminating; the formal descriptions of the older schools of academic psychology give place to a living, interesting sequence of cause and effect. The student may find the use of such terms as "dereistic," "ekphoric," "syntonic," "schizoid," etc., somewhat confusing at first, but their meaning and value are made clear. The sections dealing with treatment, both general and special, are also practical and conservative.

The task of the translator has undoubtedly been heavy, but on the whole has been successfully accomplished. There still remain many teutonisms, and the meaning of occasional sentences is obscure. It is unfortunate also that the words "case" and "patient" are frequently used wrongly, the term "organic patients" being employed many times. Words are somewhat frequently misspelled, and some are incorrectly used, as when the content of hallucinations is spoken of as being "impugnable to the patient" when the context clearly implies that they are not impugnable. Such errors are, however, of minor importance, and will doubtless be corrected in later editions. The book can be most heartily recommended, and English speaking physicians and students owe a debt of gratitude to Dr. Brill for rendering available to them this admirable work of the great Swiss psychiatrist.

RETROGASSERIAN NEUROTOMY—THE PHYSIOLOGIC AND PATHOLOGIC CONSEQUENCES. CONTRIBUTION TO THE STUDY OF NEUROPARALYTIC KERATITIS. By EDWARD HARTMANN, Formerly Resident Physician in the Hospitals of Paris. Pp. 185. Paris, Gaston Doin, 1924.

This monograph is essentially a review of the literature, mainly American, on section of the sensory root of the fifth cranial nerve for trigeminal neuralgia plus the author's personal observations on sixty-five cases which were operated on by various French surgeons.

The major part of the subject matter concerns the occurrence of of keratitis following division of the sensory root. Hartmann discusses such a keratitis as being due to a direct lesion of the ganglion, to palpebral malocclusion, to trauma of an anesthetic cornea and as occurring in the absence of any of the preceding factors and consequently presenting the characteristics of an actual trophic lesion. To grasp the significance of the author's explanation of the pathogenesis of such a keratitis, it is necessary to keep in mind the physiologic results which have been observed to follow the operation. The author has confirmed the preservation of deep sensibility in the face after section of the trigeminal nerve and accepts the facial nerve as the afferent pathway for the fibers that carry that sensation. In addition, he calls attention to the abolition of the corneal and oculocardiac reflexes, exaggeration of the vasomotor reactions on cutaneous stimulation, increased reaction of the pupil to different alkaloidal drugs and exaggeration of the sweating reaction after the injection of pilocarpin. The last three phenomena are believed to be evidence of increased excitability of the nerve terminations of the "organo-vegetative" system. It is predicated that increase in the tone of the "organo-vegetative" or parasympathetic system predisposes to decrease in the local defensive resistance of the tissues. Myosis, enophthalmos, narrowing of the palpebral fissure, reduction of intra-ocular tension and cutaneous vasodilatation are not looked on as evidence of paralysis of the sympathetic fibers but as the result of an elevation of parasympathetic tonus.

Perusal of this volume shows that the French have not yet reached American standards in the section of the sensory root for trigeminal neuralgia. The frequency of this complication in any series of cases reported by a neurologic surgeon of this country has been greatly reduced by improvement in operative technic. In many, the sympathetic fibers to the eye are closely related to the ophthalmic division and the medial aspect of the ganglion. Hence, by not disturbing the ganglion from its bed and by avoiding the medial side and the ophthalmic division, the occurrence of keratitis has been materially reduced. To say that keratitis is produced by parasympathetic stimulation rather than by sympathetic paralysis, seems to be engaging in a rather futile search for an explanation. Certainly, the impression should not obtain that postoperative keratitis occurs so frequently as to offer strong contraindication to section of the sensory root.